ARCHIVES of OPHTHALMOLOGY

Volume 3 (old series Volume 60) JANUARY, 1930 Number 1

SOME OF THE PHASES AND CONTRIBUTIONS OF AMERICAN OPHTHALMOLOGY *

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PHILADELPHIA

The onward movement of general and specialized medical achievement depends on research, clinical observation, contact with patients and teaching.

A sharp distinction between the science of observation and the science of experiment is not valid; they are partners.

The clinician and the research worker have established high standards. Each endeavors to satisfy the exacting conscience of the other. Together they strive for "an accurate and discerning application of science to meet the needs of the individual patient" (Canby Robinson); they strive, too, for the same discerning application of science to the prevention of disease, which is the outstanding tendency in contemporary medical effort.

Obviously, everywhere in the world progress is brought about by specialization and cooperation. As a distinguished English surgeon more than a quarter of a century ago wrote: "The growth of specialism is inevitable, indeed, necessary. . . . But it behooves every enlightened member of the medical profession to take care that our specialization shall be the legitimate and reasoned outcome of the growth of knowledge."

Although ophthalmology has gained and maintains a position of distinguished precision among the so-called specialties, and although its exponents are fully alive to the need of further special development, more than ever they are increasing their range of action and influence in cooperation with the great system of medicine of which their division forms an integral part. Thus each physician respects his own material, and also that of his colleagues in other departments, and each becomes familiar with the need and value of the other's work.

Naturally, cooperation between clinicians and research workers can best be secured with the help of certain agencies. Prominent among such agencies are institutes, properly physically equipped, properly

* Submitted for publication, Nov. 16, 1929.

* From the University of Pennsylvania.

* Delivered on the occasion of the dedication of the Wilmer Institute of Ophthalmology, Oct. 16, 1929.

staffed and directed, properly endowed and intimately associated with a school of medicine and its hospital, being part of a university system. In such circumstances, research, clinical observation, contact with patients and teaching find their fruitful environment.

For the purpose of dedicating an institute thus fortunately placed we meet today, and gladly participate in an outstanding event in the history of American ophthalmology.

In representative capacity, as one who comes from the oldest medical school in our country, the trustee chairman of its Board of Medical Affairs, somewhat closely concerned with its ophthalmologic endeavors—these are my credentials—and also individually, I desire, Mr. President, members of the Board of Trustees and of the faculty, to express my high appreciation of the privilege of participation in these ceremonies. I congratulate you on the consummation of your plans, and on your felicitous choice of a Director—a gifted man, well qualified by clinical observation, scientific imagination, administrative ability and teaching experience for the important duties of the position he now occupies.

The narrative of the development of European ophthalmology presented yesterday by a master of that science and of that art brings into prominence the debt, gratefully acknowledged, which the ophthalmologists of this country owe to the discoveries, instruction and stimulation of the workers in the older world.

In partial payment of this indebtedness, American ophthalmologists endeavor to be worthy beneficiaries of these exceptional opportunities so freely offered for many years.

It would seem proper, therefore, on this occasion to turn to the ledger of American ophthalmology and to examine the entries on the credit side of its pages: clinical researches and observations as such and in extension and in elaboration of others elsewhere previously made; laboratory work and the results of original investigations; the development of ophthalmologic teaching and literature, and here and there to exhibit fragments of American ophthalmologic history. The present essay is concerned with this endeavor; the selected records are woven into an unpretentious story.

ANOMALIES OF REFRACTION

Practically nothing is known about the status of ophthalmology in the colonies or during the postrevolutionary period. But it is interesting to recall that the earliest recorded contribution from this country to optical therapeutics was made by Benjamin Franklin, when he constructed, in 1784, bifocal lenses, which he described and illustrated a year later in a letter to George Whatley in London. Whether "Franklin's glasses," as they were called, came into general use in this country at that time is not a matter of record.

Now the utilization of bifocal lenses is well nigh universal. Owing to accurate calculation and ingenious methods of construction, the various types of "Kryptok" and one-piece bifocals are available, which appeal to the esthetic sense of the patients, and conceal a formerly dreaded indication of the approach, if not the arrival, of that most insidious of all diseases, old age, or, as Warthin would prefer, of "age, the major involution."

Shortly after Sir George Airy discovered and corrected the myopic astigmatism of his eye (1827), a young clergyman in New York sent to the Messrs. McAllister, opticians in Philadelphia, a highly intelligent, illustrated record of his investigation of his own visual defects, also myopic astigmatism, which enabled them to grind a lens that "materially improved his vision."

Forty-odd years later, these spectacles came into the possession of the late Dr. Henry D. Noyes.[1] On the paper in which they were wrapped, a notation in Reverend Goodrich's handwriting reads: "No. 7, French no., cylinder concave, got of McAllister, May, 1828," being a record of the first planocylindric lenses ground in this country for the correction of an astigmatic ocular defect.[2]

About 1853, Isaac Hays of Philadelphia was the earliest American ophthalmologist to prescribe cylindric lenses. His first patient, a young girl checked in her ambition to secure an education because of defective vision, was the happy beneficiary of his diagnostic acumen and of McAllister's technical skill. Ten years later, F. J. Bumstead and Hasket Derby[3] continued in this country the pioneer work of astigmatic corrections.[4]

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1. Noyes, H. D.: Am. J. M. Sc. **67**:355, 1872.

2. Isaac Hays, in his first edition of Lawrence's "Diseases of the Eye" (1843), included the Reverend Goodrich's letter to McAllister, and stated in his third edition of this book (1854) that "we have seen within the past year two cases in which this defect existed." Donders (Anomalies of Accommodation and Refraction of the Eye, translated by W. D. Moore, London, New Sydenham Society, 1864, p. 541) quoted these records in his note on the "History of our Knowledge of Regular Astigmatism," as the earliest examples of the correction of astigmatism in our country.

3. Bumstead, F. J., and Derby, Hasket: Am. Med. Times **7**:203, 1863; ibid, p. 277.

4. Curiously enough, they sent to Berlin for the glasses they ordered, because, as Bumstead said, "cylindrical glasses at present (1863) cannot be made in this country, at least in New York," and yet the McAllisters in Philadelphia had been grinding them for thirty-five years; before 1863 (exact date unknown), "spherocylinders were ground in America by the optician Zentmayer of Philadelphia, who had the distinction of being the first in this country to limit his official business to the filling of physicians' prescriptions for glasses" (Emory Hill: The American Encylopedia of Ophthalmology, vol. 7, p. 4916). "At least in New York" is a saving clause.

The credit of the next advance along these lines is due to John Green [5] of St. Louis (1867), who, were he alive today, would surely be eligible for membership in that select group of men capable of understanding the mathematical symbolizations of Einstein. Fresh from the influence of Donders, he developed his methods of the detection and measurement of astigmatism and its relation to defective vision, asthenopia and progressive myopia—methods still in use today.

A highly important phase of American ophthalmology followed. It concerned itself with the sterling results obtainable when two or more departments of the medical sciences cooperate, in this instance ophthalmology and neurology.

S. Weir Mitchell was the first American physician, not an ophthalmologist, who, collaborating with William Thomson, William F. Norris and Ezra Dyer, realized the necessity of systematic ocular examination of his patients.

Gradually the theme developed, and in 1874 Mitchell [6] published his classification of headaches from the etiologic standpoint, including as the sixth member of the group "eye-strain" as an important factor, when for the first time this convenient term, now universally employed, appears in American literature.[7]

When definitely sure of the evidence in the case accumulated by himself and his colleagues, Mitchell,[8] in 1876, wrote his famous proclamation, which reads:

> (1) What I desire, therefore, to make clear to the profession at large is that there are many headaches which are due to the disorders of the refractive or accommodative apparatus of the eyes; (2) that in those instances the brain symptom is often a most prominent and sometimes the sole prominent symptom of the eye troubles, so that while there may be no pain or sense of fatigue in the eye, the strain with which it is used may be interpreted solely by occipital and frontal headaches; (3) that the long continuance of eye troubles may be the unsuspected source of insomnia, vertigo, nausea and general failure of health; (4) that in many cases the eye trouble becomes suddenly mischievous owing to some failure of the general health, or to the increased sensitiveness of the brain from moral or mental cause.

Methods have been elaborated, instruments of precision improved and the list of interpretations of eye strain extended, but otherwise little has been added to this complete and compact presentation of the facts of the case.

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5. Green, John: Am. J. M. Sc. **53**:117, 1867; **54**:82, 1867.

6. Mitchell: Med. & Surg. Rep. **31**:827, 1874.

7. Dr. Mitchell is not the author of the term; he believed that Tyrrell, in England, first used it, but I am unable to find the reference.

8. Mitchell: Am. J. M. Sc. **71**:363, 1876.

There is not the least intention to claim the credit of an original discovery for Mitchell and his confrères; they themselves made no such claim. Ten years earlier, Donders [9] had solved the problem of asthenopia, cured patients with "nervous headaches" by the correction of ametropia and demonstrated the need of exposing latent errors of refraction. And von Helmholtz,[10] a little later, went a step further, and stated that unexplained and apparently nervous troubles may be relieved by proper glasses.

What Mitchell did was to assemble incontrovertible evidence from his own and his colleagues' large clinical experience and clinical research, to tell the story to the medical profession at large, and to indicate an avenue of therapeutic advance which in ample measure has been responsible for the relief of untold thousands, and for a large influence in fundamentally reconstructing the treatment for many functional nervous disorders.

Ophthalmic endeavor, slowly, it is true, moved from a position of isolation. Physicians began to realize not merely the necessity of specialization, which at this time was rather grudgingly acknowledged, but the importance of such specialization in medical departmental work.

A logical result of the Mitchell proclamation and of the observations which preceded it was increased accuracy in the determination of refractive anomalies and the correction of "small errors of refraction"—errors often more responsible for the evils of eye strain than those of greater degree. And the leading ophthalmologists of this country, by their teaching [11] and their practice, strove to introduce precision in refractive work as a therapeutic measure for relief from many mental and bodily disorders.[12]

Considering the methods then available—largely subjective—it is not a little astonishing how well ophthalmologists succeeded; how accurate their determination and correction of the errors of refraction, facilitated but not materially exceeded at the present time.

It is true that at about this period, indeed, a few years earlier, Ferdinand Cuiguet (1873) had introduced the shadow test (skiascopy, retinoscopy). Parent had demonstrated its optical basis (1880), and

9. Donders: On the Anomalies of Accommodation and Refraction of the Eye, translated by W. D. Moore, London, New Sydenham Society, 1864, p. 233.

10. Von Helmholtz, quoted by Hirschberg: Geschichte der Augenheilkunde, in Graefe-Sämisch: Handbuch der gesamten Augenheilk, Leipzig, Wilhelm Englemann, 1915, vol. 14, parts 5 and 7, pp. 62 and 74, par. 754.

11. William Thomson's lectures to the students in the Jefferson Medical College in Philadelphia are noteworthy.

12. Many years afterward, the late Dr. George M. Gould (Influence of Visual Function upon Health, Biograph, Clin. 1906, vol. 4) by his facile pen and insistence did much to impress the importance of this subject on the members of the profession who had not yet grasped it.

in England Lytton Forbes, A. Stanford Morton, Charnley, Henry Juler and Story had described and popularized the method.

But Edward Jackson's book (1885),[13] wherein he set forth an account of the test with a plane mirror as applicable to all varieties of ametropia, the determination being made by "measuring the variable distance of the surgeon from the patient," disclosed an original technic which attracted widespread attention and was soon followed by well nigh universal appreciation of the value of the method. Jackson's prophecy that retinoscopy would prove to be by far the most accurate test of the refraction of the eye is justified.

Many improvements in technic, types of mirrors and character of illumination have appeared, here and elsewhere, and today the determination of refractive errors is well nigh an exact mathematical procedure.

The elaborate investigations made on the continent of Europe of the influence of refractive errors on children during school life and on students in the schools of higher grade, stimulated similar studies in this country which began with Peter Callan's examinations in 1875 and were carried on by Loring, Elkanah Williams, Hasket Derby and others. Especially noteworthy is the work of the late Dr. S. D. Risley and of Dr. B. Alexander Randall, whose statistical tabulations (1885) were of great importance.

ANOMALIES OF MUSCLE BALANCE

Exterior ocular muscle imbalance was not neglected in the early studies of the so-called eye strain reflexes; indeed, Mitchell emphasized the significance of this factor as well as that which pertained to accommodative deficiency. And prior to this time von Graefe and Donders had apprehended the importance of asthenopia muscularis, as it was called.

It is a fact, however, that for a considerable period the so-called "doctrine of insufficiency" concerned itself almost solely with "insufficiency of the internal recti" as the cause of muscular asthenopia; only casual references to insufficiency of the externi are found.

The next step in advance occurred, in this country, forty-three years ago, when George T. Stevens [14] published his justly famous terminology of the heterophorias—a terminology which is everywhere accepted and universally recognized. And now the terms exophoria, esophoria, hyperphoria and cyclophoria (a term introduced by G. Price, after Giles Savage's well known studies of "insufficiencies of the obliques" were published [15]) are as familiar as the words hyperopia, myopia and astigmia.

Stevens' name is so intimately associated with this capital terminology that the danger is ever present of neglecting his highly important studies

13. Jackson, Edward: Skiascopy and Its Practical Application to the Study of Refraction, Philadelphia, Edwards, 1885.

14. Stevens, G. T.: New York M. J. **44**:624, 1886.

15. Savage: Arch. Ophth. **20**:105, 1891.

of the whole subject of muscle balance—evidence of his scholarly attainments and scientific mind. This is not an occasion on which to discuss his somewhat overenthusiastic surgical recommendations, but it is a time to emphasize the value and importance of his original and outstanding work.

Since this period and including it, the faults of ocular motility and their consequences have attracted the insistent attention of investigators and clinicians. The word "muscle-strain" appeared in the literature, and it became more and more evident that satisfactory treatment for "eye-strain," in its manifold interpretations, required not only meticulous correction of refractive errors, but also restoration of defective muscle balance to a normal standard.

Prominent among those who in America have deeply studied and contributed to this subject, Alexander Duane obtained a place in the foremost rank.

His determination of the normal range of accommodation; his classification of the various types of abnormal accommodation; his carefully thought out differentiation of the varieties of heterophoria, the methods of their detection and their symptomatology; his establishment of the essential differences between heterophoria and heterotropia; his deducations which are so largely responsible for improvements in therapeutic measures, surgical and otherwise, only imperfectly indicate the wide range of his investigations.

His chapter on "The Motor Anomalies of the Eye," [16] incorporated in his translation and edition of the world's greatest ophthalmic textbook, with characteristic excellence summarizes the results of his devotion of time and thought to the complex problems of disturbed ocular muscle balance.

It is a tragedy that death closed Duane's brilliant career before he was able to publish, although nearly completed, what was intended to be his opus majus, which contains the invaluable researches of the best years of his useful life. The hope is expressed that means may be found to rescue it and place it at the disposal of the medical profession, with the books of George Stevens,[17] G. C. Savage [18] and Lucien Howe,[19] who have devotedly labored in elucidating the problems of ocular motility.

16. Fuchs, Ernst: Textbook of Ophthalmology, authorized translation from the twelfth German edition by Alexander Duane, Philadelphia, J. B. Lippincott Company, 1924, chap. 17, p. 267.

17. Stevens, G. T.: Treatise on the Motor Apparatus of the Eyes, Embracing an Exposition of the Anomalies of Ocular Adjustments and Their Treatment, Philadelphia, F. A. Davis Company, 1906.

18. Savage, G. C.: Ophthalmic Myology. A Systematic Treatise on the Ocular Muscles, ed. 2, Nashville, Tenn., D. C. Savage, 1911.

19. Howe, Lucien: Muscles of the Eye, New York, G. P. Putnam's Sons, 1907-1908, vol. 2.

THE TOXIC AMBLYOPIAS

Among the toxic amblyopias, on which attention has been concentrated for many years, abroad and here, those caused by quinine, tobacco and alcohol (ethyl and methyl) are conspicuous.

Soon after Caventou and Pelletier isolated the alkaloid quinine, in 1820, its toxic ocular influence was recognized and reported in German and Italian literature. Although von Graefe [20] was the earliest in time to study temporary blindness caused by quinine with the ophthalmoscope, he failed to find the fundus lesions; that is, they were not present.

The first systematic investigation of such changes is due to a series of clinical studies by Voorhies,[21] Roosa [22] and by Roosa and Ely,[23] Gruening,[24] H. Knapp [25] and Michel,[26] and to them is due the description of the classic picture of the eyeground in amaurosis caused by quinine (1879-1881).

The experimental production of amblyopia with quinine began in 1847 in America with W. O. Baldwin's [27] studies, which, however, possess only historical interest. The first definite effort along these lines in our country dates from 1890,[28] when an elaborate series of researches demonstrated that toxic doses of quinine in dogs produced a replica of the picture of the fundus in human beings, an atrophy of the nerves, optic tracts and chiasm, and proved that the action of the drug on these structures is selective, other cranical nerves and the cortical areas being unaffected, and that ultimately retinal vascular changes (endovasculitis) take place, which since that time have been clinically observed in human subjects.

It is interesting to note that these experiments confirmed the results of a prior research by H. Brunner [29] in Zurich, with which the American experimenter was unfamiliar at the time.

The lesions were ascribed to the intense ischemia of the retinal vessels, and it remained for Ward Holden,[30] eight years later, to bring forward the experimental proof (1898) which indicates that quinine is directly a poison to the retinal ganglion cells, and on the

20. Von Graefe: Arch. f. Ophth. **3**:2 and 396, 1857.
21. Voorhies: Tr. A. M. A., 1879, p. 411.
22. Roosa: Arch. Ophth. **8**:1392, 1879.
23. Roosa and Ely: Arch. Ophth. **9**:41, 1880.
24. Gruening: Arch. Ophth. **10**:81, 1881.
25. Knapp, H.: Arch. Ophth. **10**:220, 1881.
26. Michel: Arch. Ophth. **10**:214, 1881.
27. Baldwin, W. O.: Am. J. M. Sc. **13**:292, 1847.
28. Tr. Coll. Phys. Phil., 1890; Tr. Am. Ophth. Soc. **6**:23, 1891-1893.
29. Brunner, H.: Ueber Chininamauroe, Inaug. Diss., Zurich, 1882.
30. Holden, Ward: Tr. Am. Ophth. Soc. **8**:405, 1897-1899.

disintegration of these cells depends the ascending degeneration of the optic nerve chiasm and optic tracts. Holden's results have been abundantly confirmed here and abroad.

From the days of Mackenzie, who in 1832 issued the first scientific statement with respect to the deleterious effect of tobacco on vision, until the present time, the attention which amblyopia caused by tobacco has received is an elaborate one.

Although there is plenty of evidence that tobacco alone, that is, unassociated with the influence of alcohol (ethyl), or possibly that one of its volatile decomposition products (as was suggested in an American contribution to this subject many years ago) is competent to produce a form of so-called central amblyopia, the exact mechanism, or the seat of the earliest lesion, is unknown.

Two facts have evolved from the accumulated evidence: 1. The scotoma produced by tobacco (or tobacco-alcohol) possesses distinctive features, as was originally portrayed by Hirschberg, shown by Groenouw in his sterling chapter on this subject, and more exactly by Traquair in Scotland, Sattler in Germany, Doyne in England and recently by perimetric researches in America (Fewell, Peter and others).

2. The amblyopia and the lesions which cause it are not due solely to tobacco (or its decomposition products), or to tobacco-alcohol, but are facilitated, in general terms, by the anemia, gastric disturbances, etc., which these agents produce, as was suggested long ago by Horner and Sachs.[31]

Direct evidence in this respect was not available until in 1903 an American research,[32] the chemical analysis of the secretions having been made by David Edsall, demonstrated in the subjects with amblyopia caused by tobacco the presence of enterogenous decomposition products, and rapid improvement when a normal metabolism was restored. Additional clinicochemical investigations appear to confirm this belief, but more are required to reach a definite conclusion.

The first case of blindness caused by methyl alcohol recognized in the United States was reported by the late J. M. Ray of Louisville, Ky., in March, 1896, to a local medical society. I have seen the manuscript of his report, which, however was not printed. The first published case is by Viger [33] in 1877, as Harold Gifford has pointed out.

Indeed, from the time when Kuhnt traced the cause of an unexplained atrophy of the optic nerve of one of his patients to the ingestion of a methyl alcohol preparation used by this man while he was an inmate of a prison in Montpellier, until the present time, the history of this toxemia

31. Sometimes retinal angiosclerosis appears to be a contributing factor (Zentmayer).

32. Tr. Am. Ophth. Soc. **10**:41, 1903-1904.

33. Viger: Ann. de méd. **2**:105, 1877.

constitutes one of the most interesting as well as the most tragic chapters in ophthalmic literature.

Casey Wood and Frank Buller's [34] important analysis, twenty-five years ago, of 153 cases of blindness and 122 deaths attributable to methyl alcohol, and S. Lewis Ziegler's [35] admirable later study of this whole subject served a sterling purpose in warning the medical profession, as well as the laity, about the grave dangers of this toxic product, often in astonishingly small quantities, and not merely as a beverage or an adulterant, but, as had been demonstrated some years earlier,[36] by inhalation and absorption through the cutaneous surface—being an ever-present menace to workers in those industries in which this agent is employed.[37]

Although Ward Holden [38] first noted nutritive changes in the ganglion cells of the retina in a dog poisoned by methyl alcohol and regarded these lesions as the cause of the blindness, a belief apparently substantiated by Birch-Hirschfeld and others abroad, these results have not been confirmed by similar researches (dogs) in the author's laboratory (Fewell and De Long [39]), nor in this city by Jonas Friedenwald and A. Felty.[40]

It may be that this contradictory evidence is caused by the kind of methyl alcohol employed, probably chemically pure in the last named instances and not so in the others. In fact, there is some evidence to show that a contamination (fusel oil [41]) is the real toxic agent (Elschnig), or perhaps that in imperfect combustion of the alcohol in the system produces formaldehyde, as Fridenberg maintained; also the blindness may be due to a direct influence of the drug on the optic nerves (Harold Gifford). (J. Friedenwald [40] believes that wood alcohol "attacks the whole optic nerve.")

The toxic action of quinine on the retinal ganglion cells appears to be definitely settled; moreover, this alkaloid itself is apparently the

34. Wood, Casey; and Buller, Frank: Tr. Sect. Ophth. A. M. A., 1904, p. 412.

35. Ziegler, S. L.: The Ocular Menace of Wood Alcohol Poisoning, J. A. M. A., **77:** 1160 (Oct. 1) 1921.

36. Ophth. Rec. **10:**289, 1901.

37. H. H. Tyson and M. J. Schoenberg experimentally demonstrated the toxic influence of methyl alcohol by inhalation, J. A. M. A. **63:**915, 1914.

38. Holden, Ward: Arch. Ophth. **28:**125, 1899.

39. The Bowman Lecture, London, Adlard & Son, Ltd., 1923, p. 83.

40. Friedenwald, J. S.: The Pathology of the Eye, New York, The Macmillan Company, 1929, p. 124.

41. Dr. Henry F. Smyth (Laboratory of Hygiene, University of Pennsylvania) informs me that his investigations render it doubtful if there is any fusel oil (amylic alcohol) in the synthetic methanols. He like Holden, found degeneration of the ganglion cells of the retinas of animals (guinea-pigs) poisoned by methyl alcohol.

poisoning agent. This is not so with tobacco and methyl alcohol (although an action on the ganglion cells cannot as yet be excluded), and it would seem that additional investigations are required to solve the problems involved. This is an inviting field for research, and should appeal to the workers in this institute.[42]

SYMPATHETIC OPHTHALMIA; UVEAL TRACT DISORDERS; FOCAL INFECTIONS

Since Mackenzie's formal description of sympathetic ophthalmia ninety years ago, this disease has been elaborately studied from all standpoints, but as yet its pathogenesis is unknown.

American contributions, not numerous but valuable, begin with Harold Gifford's [43] (1899) method of treatment with massive doses of the salicylates, being a procedure which even today, in spite of the importance which arsenical compounds have attained, is by no means dislodged from our armamentarium.

Whether Verhoeff's [44] recommendation of huge doses of diphtheria antitoxin, emphasized by reported successes, shall attract equally favorable consideration the future must decide.

Of the theories which have been advanced to explain the development of sympathetic ophthalmia, two stand forth prominently: the infective theory and the anaphylactic theory, which, as Duke-Elder has said, are rivals.[45]

According to Elschnig, its chief author, the dominant factors of the latter are: anaphylaxis of uvea on account of tissue disintegration, the chief rôle being taken by the pigment, and an anomalous condition of the organism, that is, autointoxication in the widest sense of this term.

First in the Department of Medical Research in the University of Pennsylvania (1915), and later at Johns Hopkins Medical School and Hospital, Alan C. Woods [46] undertook a somewhat detailed clinical and

42. The deleterious influence of the arsenicals, especially atoxyl and tryparsamide, on the eye is well known. An important paper as to such action by tryparsamide is one by A. C. Woods and J. E. Moore (Visual Disturbances Produced by Tryparsamide, J. A. M. A. **82**:2105 [June 28] 1924).

43. Gifford, Harold: Clinical and Pathologic Notes on Sympathetic Ophthalmia, J. A. M. A. **34**:341 (Feb. 10) 1900.

44. Verhoeff: Tr. Am. Ophth. Soc. **24**:173, 1926.

45. According to H. Gifford, the first of the biochemic theories was advanced by Brown Pusey, who suggested that the cells, probably those of the ciliary body of the injured eye, give rise to a cytoxin which, having a selective affinity for corresponding cells in the other eye, sets up an inflammation. A year later, Golovine advanced a similar theory. (The American Encyclopedia of Ophthalmology, 1920, vol. 16, p. 12409.)

46. Woods, A. C.: Tr. Sect. Ophth. A. M. A., 1917, p. 133; Arch. Ophth. **46**:503, 1917.

experimental study of Elschnig's allergic theory, which in the main corroborated his observations, which he also extended by the development of an intradermal sensitivity test, its object being to test the presence or absence of hypersensitivity to uveal pigment,[47] which has proved its value, and elaborated a form of treatment which consists in desensitization with pigment, and the continuance of the injections until a state of immunity is produced.

Woods'[48] ultimate conclusion (1925), although he fully recognized that the cause of sympathetic ophthalmia is not demonstrated, was that usually there is an allergic reaction in patients with this disease, that uveal pigment is concerned with this reaction, and that treatment with uveal pigment looking toward desensitization is of importance, but not to the exclusion of other methods usually employed.[49]

The value of the blood count in sympathetic ophthalmia as a diagnostic test of this disease, and as a sign of its imminence, has not been decided. Its importance, first urged in this country by Harry Gradle,[50] is doubted by Sanford Gifford.[51] Browning's studies in England are suggestive.

If the Elschnig-Woods anaphylactic theory of sympathetic ophthalmia is correct, removal of areas of focal infection (tonsils, teeth, intestinal tract, etc.) should be of therapeutic value, and certain observations in this respect in our country (Arnold Knapp,[52] Harbridge[53]) indicate that such may possibly be the case.

The relation of so-called enterogenous autointoxication and focal infections as etiologic factors in the development of ocular disorders, chiefly of the uveal tract, has attracted widespread attention, and in the numerous clinical and experimental researches American ophthalmologists and internists have largely participated.

From the standpoint of gastro-intestinal autointoxication, the results of an elaborate investigation, the chemical analyses having been made by Dr. Charles A. Fife, Dr. Edward Goodman and by Alonzo E. Taylor, then professor of physiological chemistry in the University of Pennsyl-

47. Dr. A. Knapp has also made clinicopathologic studies of this intradermal reaction.

48. Woods, A. C.: Tr. Ophth. Soc. U. Kingdom **45**:268, 1925 (contains bibliography).

49. Recently, S. R. Gifford and L. H. Lucic advanced reasons why they consider the theory of a virus as the cause of sympathetic ophthalmia the most plausible of the various theories (The Etiology of Sympathetic Ophthalmia, Arch. Ophth. **1**:468 [April] 1929).

50. Gradle, Harry: Ber. d. ophth., Heidelburg, 1910, p. 238; Ophth. Rec. **20**: 619, 1911.

51. Gifford, Sanford: Arch. Ophth. **47**:327, 1918.

52. Knapp, Arnold: Tr. Sect. Ophth. A. M. A., 1919, p. 242.

53. Harbridge, D. F.: Am. J. Ophth. **2**:269, 1919; **5**:791, 1922.

vania, formed the subject of an American report to the Seventeenth International Congress of Medicine in 1913.[54]

Briefly, the conclusion reached was that there is no proof that autointoxication, strictly defined as a condition in which poisons formed within the body, that is, within the metabolism, is itself responsible for ocular disorders, for instance, of the uveal tract. But as gastro-intestinal intoxication (not autointoxication) is due to the action of bacteria on the foodstuffs in the intestinal tract, these bacteria or their toxic products may be one of the causes of ocular disease, and may be, in this sense, catalogued with the focal infections resident in the teeth, tonsils, paranasal sinuses, prostate, gallbladder, pelvic organs, etc.

Focal infections in relation to the eye, so well studied in England, where, however, at the present time enthusiasm in this respect appears to be somewhat on the wane, attained in this country an especial interest after the publication of the Lane Lectures in 1916 by Frank Billings, and since that time have been elaborately investigated from the clinical, bacteriologic and experimental standpoints, especially in the Mayo Clinic by Rosenow and Benedict, and in the fine cooperative clinical researches of Irons and E. V. L. Brown,[55] and by many others who with earnest intent have recorded their clinical experiences and experimental investigations.

That the doctrine of focal infections is still widely accepted there is no doubt; that many metastatic infections in the ocular coats, chiefly in the uveal tract, are due to the bacteria which create the primary lesions, that is, the foci of infection, seems to be settled, although admittedly a complete mechanism for each organism has not been demonstrated nor have possible flaws in this etiologic relationship been eliminated. This is not the time nor the place to discuss criticism, differences of opinion or indiscreet enthusiasm. But the fact remains that in the treatment for many ocular disorders the application of the doctrine of focal infection represents a therapeutic advance and that the contributions to this subject from American ophthalmologists interpret achievements of value.

MEDICAL OPHTHALMOSCOPY

When von Helmholtz, seventy-eight years ago, introduced the ophthalmoscope to ophthalmology, a glory just missed by the unfortunately advised Babbage, the science of medical ophthalmoscopy was born, and the partnership of ophthalmic science and general medicine was established. Soon after this immortal achievement of von Helm-

54. Tr. Sect. Ophth., Seventeenth International Congress of Medicine, 1913; Pathogenesis of Chronic Uveitis, Philadelphia, Fell & Company, 1913.

55. Irons, E. E., and Brown, E. V. L.: The Etiology of Iritis, J. A. M. A. **66**:1840 (June 10) 1916; The Etiology of Iritis: Second series, ibid. **81**:1770 (Nov. 24) 1923; Tr. Sect. Ophth. A. M. A., 1926.

holtz, the ophthalmoscope was in use by American ophthalmologists, and among those who at this time made a record of their experiences, John H. Dix of Boston (1854) and Addinell Hewson and Lawrence Turnbull of Philadelphia (1855-1859) are conspicuous. At least one of them (John Dix [56]) gravely warns his readers of the dangers which surround this instrument—a caution which a year before had been sounded by James Dixon at "Moorfields" in London. They feared that "the strong light reflected by the instrument into the eye" would injure the retina. Fancy their amazement should they be able to witness a slit-lamp, a large Gullstrand ophthalmoscope or a Nordensen camera in operation.

It would be historically interesting to determine exactly who conveyed the first ophthalmoscope from Europe to America. According to Alvin Hubbell,[57] this credit belongs to Elkanah Williams. Be that as it may, Philadelphia owes its first ophthalmoscope to the late Dr. John H. Brinton, who brought it with him after a year's study in Paris in 1853.[58]

Naturally, in earlier days the lesions which the reflecting ophthalmoscope revealed in the fundus oculi, interpreting diseased processes elsewhere in the body were largely, if not entirely, within the province of ophthalmologists, and the importance of the ophthalmoscope as an instrument of precision in the investigation of general disorders was at first best revealed in hospitals and their outpatient departments.

Hence it is not without interest to note that nearly fifty years ago the late Dr. William F. Norris, as far as it was possible, directed an ophthalmoscopic examination of all patients in the medical and neurologic wards of the hospital of the University of Pennsylvania, and attached to his staff a young physician whose duty it was to see that the work was properly and systematically carried out. The enthusiasm of this youth received a stimulus which only Osler could supply when a few years later he joined the staff of that hospital, and, as you all know, after Sir William came to Johns Hopkins Hospital, he established the same kind of examination so exactly that it became an essential part of routine investigation of the patients.

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56. Dix wrote (Boston M. & S. J. **51**:436, 1854): "For the exploration of the eyes the retina of which is wholly and hopelessly insensible to light, the ophthalmoscope is of unquestionable use . . . This I conceive to be the only safe and therefore the only legitimate use to which these instruments can be applied."

57. Hubbell, A. A.: The Development of Ophthalmology in America, 1800-1870, Chicago, American Medical Association, 1908.

58. In my earlier days when I frequently assisted Dr. Brinton in his private work (he was a general surgeon of marked distinction) he often expressed his belief that the ophthalmoscope he brought with him was the first one which came to this country, certainly to Philadelphia. His son, Dr. Ward Brinton, informs me that he also often heard his father make this statement.

Coincident with the construction of the hand electric ophthalmoscope, the first one having been devised by William Dennett [59] of New York in 1885, a great increase in the employment of this instrument became apparent, and now both in hospital and in private practice its use is well nigh a matter of course.

The improvements in the modern ophthalmoscope, by no one better critically studied than by Jonas Friedenwald [60]—improvements in structure, power, sources and character of illumination,[61] and the use of lights of selected radiation—have greatly enhanced the accuracy of the investigation of the fundus, and the interpretation of the lesions portrayed.

Space does not permit even the most superficial review of the development of medical ophthalmoscopy in this country, repeating and sometimes extending observations elsewhere made, occasionally noting phenomena not previously recorded.

It must suffice, as an example, to touch on only one phase of this important, not to say entrancing, subject, namely, changes in the retinal vessels and their walls in association with hypertension and general arteriosclerosis, which have formed the subject of numerous communications in England, on the Continent and in America.

Based on a fairly accurate examination of the literature, it appears that Harry Friedenwald,[62] in association with George J. Preston, recorded the first systematic ophthalmoscopic examination in this country of patients with general arteriosclerosis, and five years later he added to his previous observations on this subject with a paper on the significance of constrictions and dilatations of the caliber of the retinal arteries in arteriosclerosis. Soon thereafter, and doubtless stimulated by Marcus Gunn's famous papers and his altogether admirable classification of these retinal lesions, American literature on this subject rapidly increased.

Gradually a catalog of suggestive signs, distinctive features and pathognomonic lesions developed. But even more important are the endeavors to differentiate ophthalmoscopically between the signs of arteriosclerosis and those due simply to hypertension, and, as Benedict has well done, to discriminate between the retinal lesions of hypertension, arteriosclerosis and nephritis.

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59. Dennett, William: Tr. Am. Ophth. Soc. **21-23**:156, 1885-1887.

60. Friedenwald, Jonas: Tr. Am. Ophth. Soc. **26**:381, 1928.

61. Early in his career, Edward Jackson (Contrib. Ophth. Sc., Jackson Birthday Volume, 1926, p. X) pointed out the advantages of direct sunlight as the source of illumination in the ophthalmoscopic examination of eyes with hazy media, and recently he perfected a technic whereby direct sunlight is available for ophthalmoscopic examination, which is highly satisfactory.

62. Friedenwald, Harry: Report on the Ophthalmoscopic Examination of Dr. Preston's Cases of Arteriosclerosis, J. A. M. A. **16**:622 (May 2) 1891; Arch. Ophth. **25**:177, 1896.

A new edition of the catalog was called for, and thanks to the work of observers here, notably H. P. Wagener,[63] whose classification is excellent, and of those abroad, especially in England, where Foster Moore's studies have acquired a high degree of merit, the result has been more than commendable.

With a reasonable accuracy, indeed, often quite sharply, it is now possible to distinguish ophthalmoscopically between the retinal vessel lesions of senility, of primary arteriosclerosis (endovasculitis), of consecutive arteriosclerosis due to general vascular hypertension and of various implantations in such eyegrounds creating complications; for instance, "retinitis of hypertension plus nephritis" (Benedict's type), and "angiosclerotic retinitis" due to local retinal vascular disease, described by Foster Moore as distinct from renal retinitis, but included by Wagener in his group of retinal changes associated with nephritis.

Recently, Wagener [64] described a new type of retinitis with malignant hypertension, usually distinguishable from other forms of vascular retinitis, and important clinical studies have been made which serve to establish a distinction between retinal angiosclerosis of purely local origin and one which interprets general vascular involvement.

These and other observations and researches, especially of our younger ophthalmic clinicians, have an honorable place on the credit page of the ledger, and again admirably illustrate the satisfying results obtained when the ophthalmologist, internist and pathologist cooperate.

OPHTHALMIC SURGERY

American contributions to surgical intervention in the eye, that is, after ophthalmology attained the rank of a definite specialty and operative work became almost exclusively the province of the ophthalmic surgeon, have been liberal in all respects. Obviously, it is impossible to do more than make reference to a few of those procedures which have earned the order of merit.

Who first in America operated for the removal of cataract is, so far as I am aware, unknown; in Boston and its vicinity this distinction belongs to Edward Reynolds. Returning from his studies in Europe in 1818, he found his father blind from cataract. "I went," he says (so his grandson reports) "to my closet and offered a prayer to deity for success, took a glass of sherry and went ahead to do my best." His prayer was answered.[65]

At an earlier date, namely, in 1808, Peter Fetter submitted to this operation at the hands of Everhard Freytag, a beloved physician who

63. Wagener, H. P.: M. Clin. North America **7**:275, 1923.
64. Wagener, H. P.: Tr. Am. Ophth. Soc. **25**:349, 1927.
65. Warren J. Collins: Boston M. & S. J. **163**:767, 1910.

practiced in Bethlehem, Pa. In his "Lebenslauf," written by his pastor and filed in the Archives of the Moravian Church, the record reads: "The operation the Lord so blessed that from that time Peter could again go around alone . . . and attend the meetings in which his heart found comfort and pasture.[66]

When Herman Knapp,[67] twenty-nine years ago, introduced his method of opening the capsule by a peripheric incision, being the second procedure of an extraction of cataract without iridectomy, it seemed as if he had largely eliminated the dangers incident to a central laceration of this membrane and for this period it was a step in advance. Next followed the revival of extraction of the lens in its capsule, whereby, whether performed according to Colonel Smith's procedure, Verhoeff's method or with the aid of any other technic, the problem of capsule it was believed by many was definitely solved.[68]

And it is interesting to recall that it should be Herman Knapp's son, Arnold Knapp,[69] who has devised in modification and cooperation an operation of this type, after subluxation of the lens (Stanculeanu-Knapp-Török method) which in the judgment of numerous surgeons ranks high among the several procedures.

But there is by no means a general agreement as to the extraction of cataract in the capsule, and to aid in reaching a decision as to which of the various operative procedures—simple extraction, combined extraction, extraction after preliminary iridectomy and intracapsular extraction —shall be the one of choice, Walter R. Parker's [70] statistical researches are unsurpassed in accuracy and effectiveness.

Among the many excellent operative procedures devised or modified by American ophthalmologists, space permits the mention of only a few of those which have secured a prominent place in ophthalmic surgery, and which bid fair to retain it: The late S. Lewis Ziegler's V-shaped iridotomy, designed to reopen a pupil occluded on account of iridocyclitis after cataract, but equally valuable in any case of after-cataract; McReynolds' pterygium operation; John M. Wheeler's original and modified blepharoplastic procedures, the result of war experience; the late Robert Reese's muscle resection operation, and the numerous modifications and improvements of muscle advancement, of partial tenotomy or tendino-expansion (Ziegler, Verhoeff, Todd, Greenwood) and of muscle recession (T. Chambers Jameson); George H. Cross' method of

66. I am indebted to Mrs. J. Upton Myers, vice president of the Moravian Historical Society of Bethlehem, Pa., for a copy of these records.

67. Knapp, Herman: Arch. Ophth. **44**:287, 1900.

68. The credit of first suggesting a method of intracapsular extraction of cataract in this country belongs to Van Hulen.

69. Knapp, Arnold: Arch. Ophth. **44**:1, 1915.

70. Parker, W. R.: Tr. Sect. Ophth. A. M. A., 1922, p. 272; ibid., 1928.

removing nonmagnetic foreign bodies from within the eyeball; many refinements in the operative procedures for glaucoma; new and modified operations for conical cornea; corneal suture in cataract operation, first suggested by H. W. Williams in 1866 (Ellett, Verhoeff). These are, as previously stated they would be, merely a few samples in the list, which could readily be extended. American ophthalmic surgery in the world's just estimation has achieved a position of honor and distinction.

RESEARCH WORK: BACTERIOLOGIC, PATHOLOGIC AND BIOCHEMICAL

Since ophthalmology came into its own fifty-odd years ago as a recognized specialized department of medicine, there is no decade in which one may not find the evidence of effort to enhance its scientific standing. To some of these efforts, from both the clinical and the laboratory standpoints, allusion has been made; others claim a brief attention.

Although mentioned by Koch, it is to John E. Weeks [71] (1886) that the credit belongs of first demonstrating by cultural and inoculation methods, that a small bacillus, now usually known as the Koch-Weeks bacillus, is a specific micro-organism for acute contagious conjunctivitis (in common parlance, "pink eye").

Although epidemics of this form of conjunctivitis have often been caused by this micro-organism, it is also true that in this country, or at least in certain sections of it, acute contagious conjunctivitis is more often due to the pneumococcus than to the Koch-Weeks bacillus, as was first demonstrated clinically and experimentally by Harold Gifford—an observation later confirmed several times by other clinicians (Veasey, Shumway and others).

That Parinaud's conjunctivitis,[72] the etiology of which was long in dispute, is due to a filamentous organism classified as leptothrix by Verhoeff,[73] who discovered it and demonstrated the characteristic histology of the lesions, is confirmed by Sanford Gifford,[74] who has also detected this organism; this is all the more interesting because it was his father, Harold Gifford, who reported the first case of Parinaud's conjunctivitis in this country.

Sanford Gifford and J. M. Patton [75] apparently have established a new form or type of conjunctivitis, named "agricultural conjunctivitis,"

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71. Weeks, J. E.: Diseases of the Eye, Philadelphia, Lea & Febiger, 1910, p. 225.

72. G. S. Derby and F. H. Verhoeff, in their studies of Parinaud's conjunctivitis, found that this disease was first described by E. Fuchs in an early edition of his famous textbook, but he gave it no name.

73. Verhoeff, F. H.: Arch. Ophth. **42**:345, 1913.

74. Gifford, Sanford: Am. J. Ophth. **10**:484, 1927.

75. Gifford, Sanford; and Patton, J. M.: Am. J. Ophth. **5**:623, 1922.

the bacteriologic content being a mixed infection with a gram-positive anaerobic bacillus as the dominant micro-organism. Sanford Gifford's study of the bacteria of chronic meibomitis, as well as the bacteriology of the normal meibomian gland, represents a satisfactory piece of laboratory work, and he and Lucic [76] not only have confirmed Szily's work on experimental transmission of uveitis from one eye to the other with herpes virus, but have given added proof of the method of transmission obtained by positive inoculations from the optic nerve and chiasm.

Whether the late Dr. Noguchi's isolation of *Bacillus folliculosis* (Noguchi) as a possible cause of trachoma in monkeys will ultimately prove that this micro-organism is also the cause of trachoma in human beings, which for centuries has baffled the research workers in all lands, is a matter for future elucidation, and in several laboratories in this country his work is being repeated and along certain lines extended.

From the standpoint of pathology, the tissues of the eye present a rich field for investigation, which has been carefully tilled by many American ophthalmologists trained in this respect, and the output is extensive.

It has always been a matter of some astonishment that many general pathologists appear to be indifferent to the opportunities presented by morbid ocular conditions—a matter which has received comment from Hofrat Fuchs. In some medical schools, for instance, in the University of Pennsylvania, in an endeavor to secure cooperation between the laboratories of general and ocular pathology, an ophthalmic pathologist has been appointed as an instructor in the laboratory of general pathology, obviously with advantages in these desirable relationships.

Some of the interesting and important contributions are: E. V. L. Brown's confirmation of Fuchs' studies of the pathologic anatomy of sympathetic ophthalmia; Verhoeff's [77] investigation of tumors of the optic nerve resulting in a demonstration that the most usual growth is glioma, awarded the Knapp Medal; also presentation of proof that tumors of the lacrimal gland are epithelial.

Holloway's [78] studies of so-called asteroid hyalitis (to which he has given the name "snow-ball opacities") and of synchisis scintillans have, he believes, settled the clinical differences; Verhoeff has demonstrated the chemical composition (recently confirmed by Fry) of the asteroid bodies.

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76. Gifford, Sanford; and Lucic, L. H.: Tr. Sect. Ophth. A. M. A., 1926, p. 20.

77. Verhoeff, F. H.: Tr. Sect. Ophth. A. M. A., 1921.

78. Holloway, T. B.: Tr. Am. Ophth. Soc. **15**:153, 1917; Arch. Ophth. **47**: 50, 1918.

A piece of cooperative research by Holloway and Verhoeff,[79] clinical and pathologic, has resulted in considerable advance in the knowledge as to the pathologic histology of gross macular disorders. Verhoeff's [80] investigation of the pathogenesis of angioid streaks in the retina indicates a possible, although not universally accepted, explanation of their nature.

Verhoeff's studies on chronic ocular tuberculosis and its experimental production; his belief that he has demonstrated, syphilis excluded, that all localized areas of choroidoretinitis are tuberculous; [81] his observation of spirochetes in syphiloma of the optic nerve (a unique experience), and his investigation with Louis Bell [82] of the pathologic effects of radiant energy on the eye—a sterling research—are examples of the activities of this accomplished research worker.

Quoting Sir William Osler, it may be said that, "a younger group of physiologic clinicians have been quietly but surely upbuilding American Medicine," and to this sentence may be added that in this membership one finds worthy ophthalmic representation.

Glaucoma, a never-ending subject of investigation and interest, which still eludes the solution of many of its problems, has naturally attracted a large share of attention. Should Jonas Friedenwald's research in the Wilmer Institute on the influence of capillary toxemia in the development of acute glaucoma, just announced at the International Congress of Ophthalmology at Amsterdam, receive confirmation, it will represent a step in advance.

Six years ago, George S. Derby,[83] with whom have been associated Prof. W. T. Boon, T. Herbert Waite, E. B. Kirk, P. A. Chandler and M. E. O'Brien, began a painstaking and carefully checked study of the light minimum and light difference in the normal and early glaucomatous eye, later modified to a determination of the curve of dark adaptation, and Derby feels that the curve of adaptation in early glaucoma furnishes information of value in a definite number of cases. An adoptometer has been constructed which is satisfactory for office use. This work, not yet completed, is one of the outstanding researches in this country in the last decade, and is surely destined to be of the utmost value.[84]

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79. Holloway, T. B., and Verhoeff, F. H.: Tr. Am. Ophth. Soc. **26**:206, 1928; Disklike Degeneration of the Macula, Arch. Ophth. **1**:219 (Feb.) 1929.

80. Verhoeff, F. H.: Tr. Sect. Ophth. A. M. A., 1928, p. 243.

81. Verhoeff, F. H.: Tr. Am. Ophth. Soc. **26**:53, 1928.

82. Verhoeff, F. H., and Bell, Louis: Pathologic Effects of Radiant Energy on the Eye, Proc. Am. Acad. Arts & Sc., 1916, vol. 51.

83. Derby, G. S.: Tr. Sect. Ophth. A. M. A., 1928, p. 37. (This article contains a review of previous papers and the literature.)

84. It is interesting to recall that Hasket Derby, George Derby's father, was the first American to employ uniform illumination of test-types (1866), and also the first American to warn against the use of mydriatics in glaucoma.

The significance which biochemistry has attained in many ocular problems finds its chief interpretation in the investigation of the elements of the crystalline lens and the development of cataract.[85]

In America, important researches are by Hektoen, and, during the last five years, in this institute by Alan C. Woods and Burky,[86] by whom the immunologic and chemical properties of the lens protein have been carefully investigated, and a mass of information on the serologic, chemical and physical properties of the crystalline body has been gathered. Much of this work is extremely suggestive. As Alan Woods pointed out, if the riddle of cataract is ever to be solved, it must be unraveled by painstaking research in the fields of biochemistry and serology, and, as he further stated, the more profound and complex the problem becomes, the more those who are inspired by scientific accuracy are convinced of the futility and danger of premature promulgation of theories and therapeutic recommendations.

The interest in research work steadily increases in our country, The scientific curiosity of the younger group grows ever keener and their efforts cover a wide range of surgery. Examples of some recent investigations are: comparative chemical studies of the ocular fluids, cerebrospinal fluids and of the blood (Martin Cohen, Kamner and Killian); cultivation of lens epithelium in vitro (D. B. Kirby, Kestry and F. Tabor); biochemical studies of the blood of patients with cataract (C. S. O'Brien); studies on the formation and chemical composition of the aqueous (Yudkin); vasomotor control of intra-ocular circulation, and the action of mydriatics and miotics on the intra-ocular blood vessels (Frank Adler); determination of retinal blood pressure and its value in the early recognition of increased intracranial pressure (Cyril Barnast, Conrad Berens); studies of the pathogenesis of papilledema (G. W. Swift); relation of the intra-ocular tension to the first appearance of papilledema (Parker); experimental ocular tuberculosis (Finnoff); roentgenologic studies of the optic canal (Goalwin).

It would be an agreeable task to make a much fuller record of the subjects chosen and the results achieved, and also to include a large number of excellent clinical studies, did time and space permit, for these, too, have a room in the house of ophthalmic interpretation. One notes with deep satisfaction how keen is the participation of the seniors

85. A. E. Davis (Tr. Internat. Cong. Ophth., Washington, 1922; Tr. Am. Ophth. Soc., 1924; Tr. Ophth. Soc. U. Kingdom **45**:186, 1925) has developed a lens antigen treatment for incipient cataract, and has reported considerable favorable response; others have failed to confirm these results (Ellis, Z. H.: Tr. Sect. Ophth. A. M. A., 1927, p. 47; New York State J. Med. **27**:1296 [Dec.] 1927).

86. Woods, A. C., and Burky: Tr. Sect. Ophth. A. M. A., 1927. (This article contains a review of previous observations and of the literature.)

in this labor, not always necessarily as actual experimenters, but ready with suggestions, inspiration and help.

NEUROLOGIC PERIMETRY

The development of ophthalmic neurology and particularly of the localization of intracranial lesions has made surprising advances during the last twenty years, not a little aided by exact visual field observations, according to the technic of quantitative perimetry, which, thanks to Bjerrum, Rönne, Traquair and others abroad, and as is not generally known, at an early date to Edward Jackson in this country, has so greatly improved the accuracy of these investigations.

Today perimetry has acquired the status of a fine art, nowhere better displayed than in the rich clinical services of Harvey Cushing, with the aid of his associates, among whom Clifford B. Walker,[87] by his technical skill and original work, has achieved a reputation of notable distinction.

Here Dandy can testify to the help offered by exact field charting admirably provided for in the Wilmer Institute and from Frazier in the neurosurgical department of the University of Pennsylvania, similar testimony is available, where in addition to his own staff work the laboratory of perimetry established and directed by Holloway, ample in its equipment and technical advantages, is at his disposal with Miss Wentworth as the accomplished perimetrist. The same is true of the other neurosurgical departments in our country, where it is fully realized that visual field testing is a method of precision in the study of localization of intracranial tumors of paramount importance, and requires and receives a technic as exact as that of the surgical procedure contemplated.

It is manifestly impossible on this occasion to attempt, even most superficially, a review of the researches encompassed in this subject. The studies of the types of hemianopia due to disorders of the pituitary body (Cushing and Walker); the rôle and disposition of the scotomas caused by hypophyseal disease, first emphasized and extended (but not first described) by investigators in this country; the hemianopias due to neoplasm of the temporal lobe (Cushing), and in contrast those due to tumors of the lateral part of the transverse fissure (W. T. Lillie); the chiasmal syndrome of primary optic atrophy and bitemporal field defects in adults with a normal sella turcica (Cushing and Eisenhardt), and the distortion of the field created by pressure of tense basal vessels on surrounding nerve structures, so well indicated as possible by John Parsons Schaeffer's [88] admirable dissections, would fill a chapter.

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87. Walker, C. B.: Brain **37**:341, 1915; Arch. Ophth. **44**:369, 1915.

88. The Bowman Lecture, London, Adlard & Son, Ltd., 1923; Tr. Ophth. Soc. U. Kingdom, 1923, vol. 43.

One always strives for increased accuracy, and in this institute Ferree and Rand, adding to many previous researches, are carrying out an ambitious program with respect to the science of perimetry: studies of the characteristics of the visual response from the center of the periphery of the retina; studies of factors which cause variability of results in testing these responses; studies of the distribution of sensitivity to light and color over the retina; studies of diseases of the eye in relation to test results, hoping ultimately to show the effects of various pathologic lesions in the test results and their significance in relation to diagnosis and prognosis.[89]

SOME THERAPEUTIC, DIAGNOSTIC AND OTHER CONTRIBUTIONS[90]

In this country, A. E. Prince first called attention to the great value of chauffage without cauterization, pasteurization, as he called it, in the treatment for corneal ulcer, and developed a special technic, which was a real step in advance, and when W. E. Shahan[91] devised the thermophore, whereby the degree of heat can be exactly determined, an admirable instrument became available, which has met many indications other than those for which it was originally designed.

Always keenly interested in therapeutic advance, ophthalmologists have not failed to realize the growing importance of ultraviolet therapy, so well indicated in England and on the Continent, and most advantageously used in the treatment for chronic ocular inflammations, especially those of tuberculous origin.

Although there is considerable difference of opinion about nonspecific or foreign protein therapy (and this is not the occasion on which to analyze the evidence, or discuss the comparative worth of the various proteins, or the really active principles of the media), there is no doubt that its utilization, properly controlled, has in numerous instances achieved remarkable results.

Among the many communications on this subject are: Hunter Scarlett's[92] experimental research on the comparative value of the agents employed; Harvey J. Howard's[93] investigations of the intravenous use of typhoid—paratyphoid vaccine, antedated by W. H. Wilder's similar

89. Point perimetry enthusiastically pursued by John N. Evans according to a specially devised method of his design, and named "angioscotometry," represents an interesting research (Brit. J. Ophth. **77**:369, 1927).

90. Samuel Theobald in this country, being unfamiliar with its previous use abroad by Continental ophthalmologists (von Graefe, Just and others), introduced boric acid into ophthalmic practice (New York M. Rec. **17**:140, 1880).

91. Shahan, W. E.: Tr. Sect. Ophth. A. M. A., 1916, p. 298.

92. Scarlett, Hunter: Am. J. Ophth. **10**:747, 1927.

93. Howard, H. J.: Tr. Am. Ophth. Soc. **26**:275, 1928.

observations and Ben Witt Key's [94] elaborate studies of this problem chiefly with respect to antidiphtheritic serum as the agent.

Arnold Knapp's studies of the pupil response to epinephrine in glaucomatous eyes (also investigated by Schoenberg and others) are important, as are Ellett's critical clinical investigations of the value of an epinephrine substitute (glaucosan) in the treatment for iritis.

Although the detection of metallic foreign bodies within the eyeball by means of roentgen investigation was an accomplishment not long after Roentgen's immortal discovery in 1895, the exact localization of the intra-ocular fragment was another matter.

In 1897, the late Dr. William M. Sweet [95] first described his well known method of localizing foreign bodies, and designed his localizing apparatus, which subsequently was greatly improved as to its structure and convenience of use and technic, and he rightfully achieved widespread reputation. Similar good fortune attended the work along these lines of the accomplished radiologist of the New York Eye and Ear Infirmary, Dr. George Dixon.[96]

Reference has been made to some of the phases of ophthalmic work incident to the necessities of the World War. Among those as yet unmentioned preeminently are the vitally important studies, experimental and otherwise, from the ocular standpoint, with respect to aviation and the selection of aviators, for which ophthalmologists are so largely indebted to the director of this institute and to those who directly or indirectly worked with him, and after him, here and abroad, along these lines (Lancaster, Berens, Howard, Cobb and others [97]).

Although the field of comparative ophthalmology has not been tilled extensively by American ophthalmologists, to the everlasting credit of the tireless worker, Casey A. Wood, ophthalmologists owe a superbly illustrated investigation of the fundus oculi of birds, as viewed by the ophthalmoscope, being a study in comparative anatomy and physiology of the highest importance. Recently, Alan Woods and Chesney reported at the International Congress of Ophthalmology in Amsterdam the isolation of a filtrable agent in the periodic ophthalmia of horses, and they have possibly determined the etiologic factor.

In the record of an earlier research, presented by Woods and Burky to the American Ophthalmological Society in July, 1929, they were unable to confirm the conclusion reached by E. C. Rosenow and F.

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94. Key, Ben Witt: Tr. Am. Ophth. Soc. **17**:470, 1919.

95. Sweet, W. M.: Tr. Am. Ophth. Soc. **8**:91, 1897-1898.

96. Max Stern, of the Philadelphia Polyclinic, made the first attempt at localization of intra-ocular foreign bodies in Philadelphia; his method, although inexact, in several instances was eminently successful.

97. Wilmer, Berens, Lancaster, Howard and Cobb: Tr. Am. Ophth. Soc. **17**:171, 186, 195 and 249, 1919.

Park Lewis [98] that a bacillus which these observers have isolated, and which they have named *Flavobacterium ophthalmicae,* has etiologic significance in periodic ophthalmia of horses. To A. Prangen [99] ophthalmologists owe a good study of the comparative anatomy of the extraocular muscles.

PUBLIC EYE SERVICES; OPHTHALMIC TEACHING; OPHTHALMIC LITERATURE

The story that I have endeavored to tell properly closes with reference to the establishment of public eye services, the opportunities of ophthalmic instruction and the contributions to ophthalomologic literature in America.

Although Elisha North's reputation rests chiefly on his work and writings as a general practitioner, he was greatly interested in diseases of the eyes. Wishing to extend his influence in this respect for the benefit of the public and to inspire others elsewhere to do likewise, he founded the first Eye Infirmary in this country, in New London, Conn., in 1817.

Three years later Edward Delafield and J. Kearney Rogers obtained two rooms in the second story of 4 South Chatham Street, New York, "as an Eye Infirmary for the poor," which in the following year was publicly opened as the New York Eye and Ear Infirmary.

In the same year and the following one, George McClellan's "Institute for Diseases of the Eye and Ear," in Philadelphia, and the "Pennsylvania Infirmary," for the same purpose, came into existence, and one may shrewdly surmise that rivalry was not lacking. Their period of activity was short. The Baltimore Dispensary was founded in 1823, and George Frick was made director of its four wards.

Should a visitor stand in Charles Street, as visitors so often do, and gaze with admiration at the Massachusetts Charitable Eye and Ear Infirmary, founded by Edward Reynolds and John Jeffries, it might well be difficult for him to realize that these stately buildings had their modest origin in a few small rooms one hundred and five years ago.

Thanks to a generous legacy of James Wills, the Wills Eye Hospital in Philadelphia was founded in 1832, and was opened for active work two years later, with Isaac Parrish, George Fox, Isaac Hays and Squier Littel as attending surgeons, the last two with George Frick of Baltimore being usually regarded as the pioneers of American ophthalmology.

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98. Rosenow, E. C., and Lewis, F. P.: Tr. Sect. Ophth. A. M. A., 1928, p. 30.
99. Prangen, A.: Tr. Am. Ophth. Soc. **26**:353, 1928.

Public service in the cause of ophthalmology thus begun has steadily increased, and today, independent of special hospitals and infirmaries, there are few hospitals of importance in this country which are not equipped with a department devoted to the treatment for disorders and injuries of the eyes.

Although instruction in ophthalmology was given as early as 1822, in the Maryland Hospital by George Frick, in the Massachusetts Eye and Ear Infirmary in 1834 by Edward Reynolds and John Jeffries, and although Henry W. Williams organized a class of Harvard students in 1850, it was not until 1860 that Miami Medical College established the first chair of ophthalmology in this country, with Elkanah Williams as its incumbent.

A happy selection indeed; he was a gifted man, an impressive teacher, vigorous in mind and in body, of great personal charm, and so skilful that of him it has been said "though love is blind, he could make love see."

In 1868, Henry D. Noyes was elected professor of ophthalmology in Bellevue Medical College, and three years later Henry Williams, advanced from a lectureship he had previously held, became professor of ophthalmology in Harvard, and ultimately endowed the chair which bears his honored name.

In 1872, Dr. William F. Norris was elected clinical professor of ophthalomology in the school of medicine at the University of Pennsylvania (later, in 1891, he was made full professor with a seat in the major Faculty), and for the fourth time ophthalmology attained its proper place in the departmental medical and surgical instruction in this country, which it now retains in all class A medical schools.

Although the instruction thus obtained abundantly suffices for the student in undergraduate years, should he contemplate adopting ophthalmology as a specialty in his future career, postgraduate work becomes essential.

Happily, the short, so-called "polyclinic" courses, wholly inadequate in this respect, are rapidly disappearing, and the opportunity for postgraduate education in this country has made, and is making, highly noteworthy advances.

When a graduate school of medicine, including a department of ophthalmology, is part of a university system, so organized that it takes advantage of all other available or required medical and scientific instruction in hospitals, laboratories and industrial plants, a comprehensive plan of graduate ophthalmologic education is possible, leading after one year or more of work to a certificate or suitable degree. Such opportunities in this country are constantly increasing, and it is not improper to indicate, as a noteworthy example of success along these lines, the graduate school of the University of Pennsylvania, equipped

not merely with its own physical properties, but also by extensive affiliation with hospitals and laboratories within its environment.

The summer school of ophthalmology, established and guided by Edward Jackson, has demonstrated its large value for ophthalmologists who desire intensive training, the instruction being by teachers especially equipped to undertake the subjects assigned. And a somewhat similar effort under the guidance of the Academy of Ophthalmology and Otolaryngology is made available to the satisfaction of those who in ever-increasing numbers attend its periods of instruction.

Since May, 1917, the American Board for Ophthalmic Examinations has been in operation, guided by its efficient secretary, W. H. Wilder, its chief and highly important function being to establish a standard of fitness to practice ophthalmology and to conduct examinations to test the qualifications of those who are practicing, or intend to practice ophthalmology.

The certificate of this board is a sine qua non of application for membership into the two most important ophthalmic societies of our country. It may be regarded as the hall mark of a qualified ophthalmic practitioner.

During the century which has elapsed since George Frick published the first American "Textbook of Ophthalmology" (1823), which won high commendation here and in England, the product of such volumes of American authorship has been large, and in the main these volumes have justified their existence, served a useful educational purpose and compare favorably with similar books elsewhere published.

The most important American work of composite authorship is "A System of Diseases of the Eye by American and Foreign Authors," edited by W. F. Norris and Charles A. Oliver (1897-1900). It remains today an authoritative interpretation of the ophthalmic knowledge of the period when it was published.

The eighteen volumes of "The American Encyclopedia of Ophthalmology" are a treasure house of ophthalmic information and a monument to the indomitable energy and fine scholarship of the accomplished editor, Casey A. Wood, who is also editorially responsible for "A System of Ophthalmic Therapeutics," (1909) and "A System of Ophthalmic Observations" (1911), books of composite authorship.

The Ophthalmic Year Book, edited by Edward Jackson and his various associates, most satisfactorily kept its readers in touch with foreign ophthalmic literature, and its discontinuance is greatly regretted.

Robert von der Heydt's "Translation of Vogt's Atlas of Slit-Lamp Microscopy of the Living Eye" served its highly useful purpose of introducing widely in America the science and art of biomicroscopy.

Arnold Knapp's "Medical Ophthalmology" (1918), which portrays the author's happy gift of lucid condensation, and "The Eye and the

Nervous Sytem," a volume of composite authorship, edited by Posey and Spiller (1908), are the chief books on these subjects of American authorship; each should appear in a new edition incorporating fresh information from the wealth of material which has accumulated since its publication.

The illustrations in Arthur Bedell's recent "Atlas of Photographs of the Fundus Oculi" have no superior. One wishes that Henry D. Noyes could scan these well executed plates. It was he who nearly seventy years ago made in America the first attempt to photograph the fundus oculi.

Alfred Cowan has performed a real educational service in the publication of his recent manual on "Physiologic Optics," being the outcome of his lectures to the students of the Graduate School of Medicine of the University of Pennsylvania, whereby with the aid of his original drawings and simplified text, he has definitely lessened the difficulties of this important subject, and Luther C. Peter's "Principles and Technic of Perimetry" (two editions) has met a deservedly favorable reception, indicating the ever-increasing interest in this essential method of ocular investigation. Török and Grout's "Surgery of the Eye" (two editions) has been well received, and Charles May's "Manual of Diseases of the Eye," intended for students, has been most successful, as its numerous editions attest; also Thorington's books on "Refraction" and the "Shadow Test."

The ARCHIVES OF OPHTHALMOLOGY,[100] familiarly known as *"Knapp's Archives,"* founded sixty years ago by Herman Knapp, represents an outstanding event in the history of American ophthalmology. Edited by him for many years, succeeded in this capacity by his son Arnold assisted by Ward Holden, and now in charge of an editorial board composed of Arnold Knapp, William Zentmayer, George S. Derby, Sanford Gifford, Francis Adler and J. Herbert Waite, this journal has always held, and continues to hold, a place second to none in this country, and compares favorably with the best ophthalmic journals of Europe.

In 1918, a new *"American Journal of Ophthalmology"* was founded, with Edward Jackson as editor. During Jackson's incumbency as active editor (ably succeeded by W. C. Crisp), his happy faculty of careful appraisement of clinical observations, of laboratory results and of therapeutic recommendations was evident in a series of initialed editorials

100. Other ophthalmologic journals were: Alt's "American Journal of Ophthalmology (1884); "Ophthalmic Record," edited by Dr. Giles Savage (1891); "Annals of Ophthalmology" (founded by Dr. J. P. Parker, 1892, and later editorially supervised by C. Wood, H. Wurdemann, W. T. Shoemaker and C. W. Parke), and "Ophthalmology" (Wurdemann, 1904).

which have exercised a notable influence in the advancement of ophthalmic knowledge in this country.

Even as *"Knapp's Archives"* constitutes the keystone of American ophthalmologic journalism, and is the repository of the best efforts of American scientific ophthalmic publications, so the American Ophthalmological Society represents the foundation of American ophthalmic associations.

Inspired by Hasket Derby, Henry D. Noyes and F. T. Bumstead, this society was established on June 7, 1864, with Edward Delafield as its first president. Founded with the high purpose of elevating the standards of ophthalmic scientific issues, and keeping pure the methods of ophthalmic practice, the hope is confidently expressed that it shall never forsake its lofty aim nor forget the dignity with which it was endowed by its distinguished originators.

Since the foundation in 1878 of the Ophthalmic Section of the American Medical Association, in 1895 of the Academy of Ophthalmology and Otolaryngology, and of numerous ophthalmic sections and societies in all of the principal cities of this country, American ophthalmic literature has reached a sturdy growth, and has gained a position of importance recognized in the published writings of the older world.

The influence of American ophthalmology is not confined entirely to America. It is gratifying to realize that students from the older world are beginning to come to our country to avail themselves of opportunities offered.

In far-off China, largely through the efforts of Dr. Harvey J. Howard, covering a period of fifteen years, first at Canton in the University of Pennsylvania Medical School, and later and for a longer period at Peiping in the Union Medical College, groups of Chinese have been intensively trained for important posts as the heads or subheads of ophthalmic departments, which they now occupy or have occupied in various medical schools. In addition, more than 150 Chinese and foreign physicians have completed postgraduate courses in the Union Medical College in Peiping under Dr. Howard's direction and are successfully laboring in the large field of ophthalmic endeavor which China presents. This is an accomplishment of which American ophthalmology may justly be proud.

Thanks to the scholarly researches of men like Harry Friedenwald, Casey Wood, Alvin Hubbell, Thomas Shastid, Samuel Horton Brown, Emory Hill, Thomas Moores Ball, Burton Chance, Fielding Garrison and many others, the history of ophthalmology from earliest times to the present period has not been neglected.

Ophthalmologists contemplate with deep satisfaction the progress of events, contemplate with enthusiasm the workers in the newly established institutes, here, at Harvard, St. Louis and Chicago, and in specially

equipped and endowed laboratories in which the workers are giving fine example of the conduct of the higher ophthalmic life in their original investigations. They are profoundly grateful to those who of their generosity have made possible these advances in scientific activities.

I have endeavored, imperfectly, to attract attention to a sort of "rudimentary map" of a few of the phases of American ophthalmology during the last century. Many of the tracings are incomplete; many are lacking.

Would that it had been possible to make fuller record and more frequent direct mention of those who have striven and are striving worthily in the ophthalmic vineyard! For all those who are banded together for the advancement of ophthalmology, here and elsewhere, I express the hope that their endeavors may continue to meet an abundant success.

A PORTABLE ADAPTOMETER *

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AND

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An apparatus for testing both the light minimum and the light difference in dark adaptation and the use of this apparatus in the early diagnosis of glaucoma have been described in previous publications by Bovie;[1] by Waite, one of us (G. S. D.) and Kirk,[2] and by two of us (G. S. D. and P. A. C.) and O'Brien.[3] In the last of this series of papers, it was shown that a test of the light minimum is of greater value in the diagnosis of glaucoma than is a test of the light difference. Since, therefore, only the light minimum need be tested, it becomes possible to devise an instrument simpler in construction than the one previously described, which may be used in the ordinary dark room of a physician's office.

NECESSARY CHARACTERISTICS OF INSTRUMENT

The essential and desirable characteristics of an instrument by means of which accurate determinations of the light threshold may be made during the course of dark adaptation may be summarized as follows:

Standardization of Scale.—In order that determinations made at widely separated intervals of time may be accurately comparable, the instrument must be so standardized that the same scale reading corresponds at all times to the same brightness of the test spot. The

* Submitted for publication, July 3, 1929.

* From the Ophthalmological Department, Harvard Medical School, and the Massachusetts Eye and Ear Infirmary.

* Read at a meeting of the American Ophthalmological Society, Hot Springs, Va., June 11, 1929.

1. Bovie, W. I.: An Apparatus for Clinical Research on Vision, Annual Report of Conservation Bureau, City of Boston, City Document 9A, 1924, p. 15.

2. Waite, J. H.; Derby, G. S., and Kirk, E. B.: The Light Sense in Early Glaucoma, Particularly the Achromatic Scotopic Threshold at the Macula, Tr. Ophth. Soc. U. Kingdom **45**:301, 1925. Derby, G. S.; Waite, J. H., and Kirk, E. B.: Further Studies on the Light Sense in Early Glaucoma, Arch. Ophth. **55**:575, 1926.

3. Derby, G. S.; Chandler, P. A., and O'Brien, M. E.: Further Studies on the Light Sense in Early Glaucoma; The Smallest Difference in Brightness Perceptible to the Light Adapted Eye (Light Difference), Tr. Sect. Ophth., A. M. A., 1928, p. 37.

brightness should, moreover, be expressed in absolute units if the results obtained with different types of adaptometers are to be compared.

Range of Brightness.—In previous experiments [2] in which the course of dark adaptation was determined for normal observers, for patients with early glaucoma and for those with established glaucoma who have and have not been operated on, it was found essential that the apparatus used be capable of furnishing a wide range of variation in the brightness of the test spot. Several factors are responsible for this. The magnitude of the light threshold for a given person at the beginning of dark adaptation is frequently as much as 100 times its value after thirty minutes or more of dark adaptation. The variation, moreover, in the light thresholds of different observers is considerable, even for normal eyes. When, however, the group of observers to be tested includes not only normal observers but those with pathologic changes in whom the disease conditions are in various stages of advancement or recession, the variation is still greater. A patient with established glaucoma may, for example, require 100 times as much light for a threshold sensation as is required by a normal observer after the same interval of dark adaptation. Difference in size of the pupil is a third cause of variation in the value of the light threshold, which becomes of especial importance if the procedure recommended by us, of immobilizing the pupil with pilocarpine, is followed.

It has been pointed out in previous articles that in order to use the value of the light threshold in diagnosis either the influence on the results of the size of the patient's pupil must be controlled or else a correction must be applied in making the computations. Our procedure consists in immobilizing the pupil, measuring its diameter and applying a correction factor to the results in order to eliminate the effect of this variable. Since the area of the pupil after it has been fixed with pilocarpine may be as small as 1 sq. mm., whereas after an iridectomy it may easily be as great as 50 sq. mm., there may result from this cause alone a fifty fold variation in the light threshold. Our previous experience has shown that, due to the various factors just outlined, an instrument for measuring the changes in the light minimum during dark adaptation should be capable of a variation in intensity between the approximate limits of 1 and 30,000 micromillilamberts.

Ease of Operation.—It is desirable, although perhaps not essential, that the brightness of the test spot be continuously adjustable by a single regulating device. A means of recording automatically each measurement of the light threshold is also of decided advantage in view of the fact that all the determinations must be carried out in the dark room.

Data on Normal Observers.—If the measurements obtained with the adaptometer are to be used in the detection of pathologic conditions

of the eye, obviously one must first have considerable information as to the results to be expected from normal observers. It is not sufficient to have data on the average dark adaptation curve for a group of normal observers. This represents merely the central tendency of the group and gives no indication of the extent to which a given normal person may differ from the average. If, however, a statistical study is made of the results obtained by testing a sufficiently large group of normal observers, it becomes possible to set a limit to the probable extent of normal variation. The results for any given person which fall outside this boundary are to be regarded as indicative of possible pathologic changes. The greater the deviation from normal, the more certain one may be of the existence of a pathologic condition.

OTHER ADAPTOMETERS USED IN CLINICAL MEASUREMENTS OF LIGHT SENSE

Before proceeding to a description of our own apparatus, we shall discuss briefly several forms of adaptometer which have been devised by others. We shall not attempt to give here a complete account of all the devices which have been used in testing the light threshold. A description of the instruments devised by Nagel,[4] Piper[5] and Ferree and Rand[6] should, however, be of interest for comparison with the description of our apparatus which follows:

In Nagel's adaptometer,[4] light from three incandescent lamps is transmitted through two milk glass plates to a third, which serves as the test spot. The brightness of the third plate is regulated by varying the illuminated areas of the other two surfaces. An Aubert diaphragm placed directly behind one of the milk glasses provides for the fine changes in brightness. The area of the diaphragm may be varied from 1 to 10,000 sq. mm. The gross changes in brightness are produced by inserting screens containing a series of holes behind the other milk glass plate. Three screens are provided, each of which reduces the effective area of the luminous surface to 1/20. The three screens together provide for a reduction in brightness to 1/8000 of the original value. The ratio of the highest to the lowest brightness is therefore 80,000,000:1. Since the maximum brightness of the test spot is equivalent to an illumination of 1.45 meter candles, the absolute level

4. Nagel, W. A.: Zwei Apparate für die augenärztliche Functionprüfung, Ztschr. f. Augenh. **17**:201, 1907.

5. Piper, H.: Zur messenden Untersuchung und zur Theorie der Hell-Dunkel-adaptation, Klin. Monatsbl. f. Augenh. **45**:357, 1907.

6. Ferree, C. E., and Rand, G.: Lantern and Apparatus for Testing the Light Sense and for Determining Acuity at Low Illuminations, Am. J. Ophth. **3**:1, 1920. An Acuity Apparatus with Attachments for Testing the Light and Color Sense and for Color Mixing, Arch. Ophth. **55**:245, 1926.

and the range are such that the instrument is suitable for all possible cases.

An improved form of the Nagel adaptometer has been devised by Piper,[5] in which two Aubert diaphragms are substituted for the single diaphragm used in conjunction with screens containing fixed openings. As a result of this modification, it is possible to vary the brightness of the test spot in a continuous series from the highest to the lowest point by first decreasing one diaphragm to its smallest area and then decreasing the second in the same way. According to Piper, it is unpractical to use apertures smaller in area than 16 sq. mm., because when this point is reached, it is difficult to make a sufficiently gradual change in brightness. Since the area of each diaphragm when completely opened is 10,000 sq. mm., the range of intensity provided is slightly less than 400,000:1. No provisions are made in either of these instruments for controlling the voltage at which the incandescent lamps are operated.

Ferree and Rand[6] pointed out the difficulty of illuminating uniformly a surface of the size needed in testing the light threshold. In their apparatus, this difficulty is overcome by employing a surface so small that the desired evenness in brightness is easily obtained. A magnified image of this surface is then projected on a larger surface at a distance of 6 meters by means of a suitable lens. The use of a projection lens also solves the problem of providing a means of varying the brightness of the test spot without altering the evenness of distribution of the light or changing the size or shape of the image. The principle employed in accomplishing this is well illustrated in the eye. The brightness of the retinal image is regulated by means of the pupil without producing any change in the shape, size or evenness of illumination of this image. For, since each point in the image receives light from the entire lens, any reduction in the effective aperture of the lens produces an equal reduction in the amount of light reaching each point in the image. The brightness of every part of the image is therefore proportional to the exposed area of the lens. In Ferree and Rand's apparatus, the reduction in the effective area of the lens is accomplished by placing a variable diaphragm as close as possible to the projection lens. No data are given as to the range of intensity obtainable with this instrument. Fluctuations in light intensity due to changes in current are controlled by means of an ammeter and a rheostat.

In the instruments described, variable diaphragms have been used in each case to change the brightness of the test surface. In other forms of adaptometers, changes in the distance of the light source, Nicol prisms, and absorbing wedges are some of the methods used. The range which may be conveniently obtained by varying the distance of the source is limited. The intensity of light transmitted by a pair of Nicol

prisms varies as the square of the cosine of the angle between them. It is therefore zero when the prisms are at right angles, increases continuously as the angle between them is decreased and reaches its maximum when the angle is zero. Consequently, it is possible theoretically to obtain intensities ranging between any desired limits by choosing a light source of the proper candle power. In order to obtain, for example, intensities ranging from 30,000 to 1 micromillilamberts the source of light must be so chosen that the intensity of light transmitted by the Nicol prisms is 30,000 micromillilamberts when the angle between them is zero. Intensities of 3,000, 300, 30, and 3 micromillilamberts correspond then, respectively, to angles of 71, 84, 88, and 89.5 degrees, approximately. It is obvious from these figures that a high degree of precision cannot be obtained in the variation of the intensity at the low end of the scale. This difficulty could be avoided by the

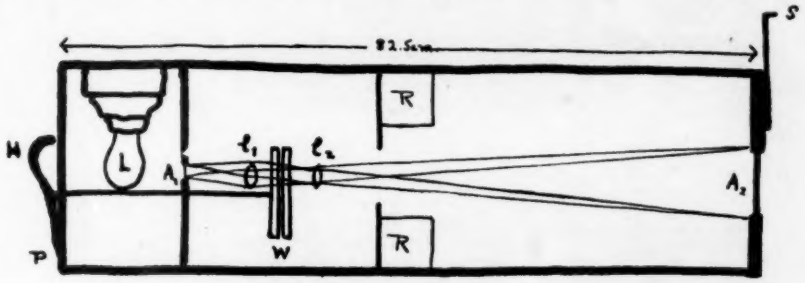

Fig. 1.—Drawing of apparatus. *L*, source of light—15 watt Mazda lamp. A_1 circular aperture, diameter 1.3 cm., covered with ground glass; l_1, 13 diopter lens, at focal distance from A_1; l_2 2 diopter lens, forming magnified image of A_1 at A_2; A_2, circular aperture, diameter 7.5 cm., covered with milk glass; *W*, paired photographic wedges for varying intensity of image at A_2; *H*, handle to operate wedges; *P*, pointer to indicate on scale the position of wedges; *S*, shutter for covering aperture A_2; R, voltage regulator—maintains constant voltage at lamp terminals.

use of auxiliary means of reducing the intensity in fixed steps. The convenience of a single adjustment for varying the intensity would, however, be lost.

In each of the two remaining methods, the use of a variable diaphragm and of absorbing wedges, certain advantages and disadvantages may be pointed out. A diaphragm is probably simpler and more likely to remain in proper adjustment than a wedge. The range of variation provided, however, is determined by the ratio of its largest to its smallest aperture. Unless large diaphragms are used therefore, it must be possible to decrease the aperture to an extremely small value in order to obtain an adequate range. In this case, especial care must be taken that at low intensities a sufficiently fine adjustment is pro-

vided. Absorbing wedges, on the contrary, may easily be constructed of any desired range of transmission. Moreover, if the density of the wedge is made proportional to the distance along its length, the percentage change in brightness corresponding to a given amount of movement of the wedges is the same at every point. For these reasons, wedges have been used to vary the intensity in the instrument we are about to describe.

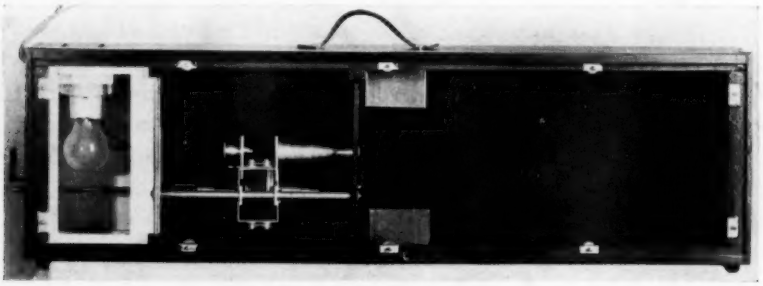

Fig. 2.—Full length side view of apparatus.

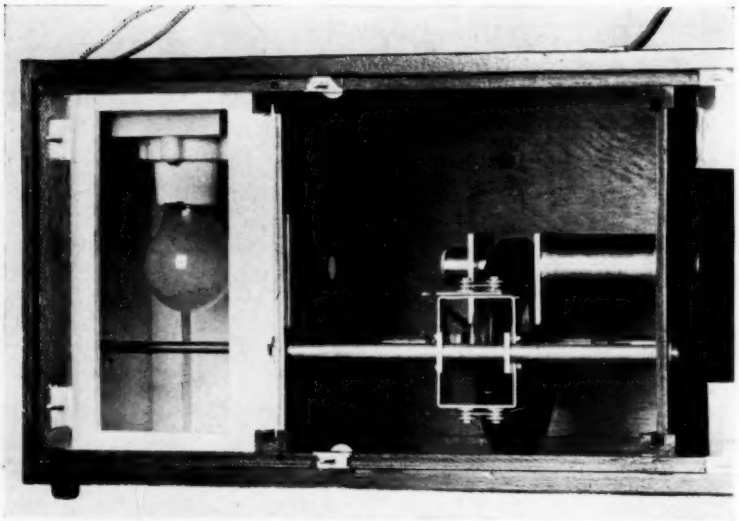

Fig. 3.—Side view of apparatus, showing lamp compartment and wedges.

DESCRIPTION OF APPARATUS

Projection System.—The apparatus is shown diagramatically in figure 1. Photographs of different views of the apparatus are shown in figures 2, 3, 4 and 5. L, the source of light, is a 15 watt Mazda lamp frosted inside. This lamp illuminates a circular aperture A_1, 13 mm. in diameter, covered with ground glass. The frosted surface of the lamp bulb and the ground glass covering of the aperture serve to produce an evenly illuminated surface. As a further aid in

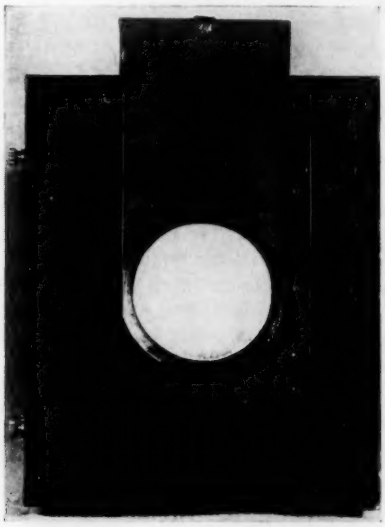

Fig. 4.—End view of apparatus, showing test spot.

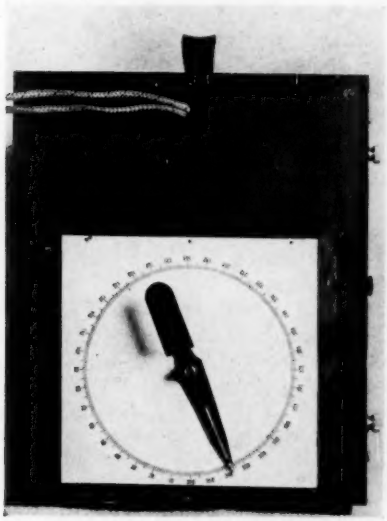

Fig. 5.—View of apparatus showing pointer and recording scale.

obtaining an even illumination, the inside walls of the compartment containing the lamp are covered with a mat of white paint. Two lenses, l_1 and l_2, form a magnified image of aperture A_1 at A_2. The light rays diverging from a point on the surface of A are rendered parallel by l_1, a 13 diopter convex lens placed at a distance equal to its focal length (77 mm.) from A_1. This parallel beam of rays falls on l_2, a 2 diopter convex lens, and is brought to a focus at A_2, 500 mm. from l_2. There is formed, therefore, at A_2 a magnified image of A_1. Since the diameters of object and image are in the ratio of 500:77, the circular luminous spot projected at A_2 by this lens system has a diameter of 84.5 mm. The aperture at A_2 is, however, made smaller in order to obtain a spot with sharply defined edges. The aperture is covered with single flashed opal glass which serves to diffuse the light falling on it and thus to render the brightness of the spot independent of the angle at which it is viewed. To the patient seated at a distance of 130 cm. from the spot, it subtends a visual angle of 3.3 degrees.

Control of Light Source.—In order that the intensity of light delivered by the Mazda lamp may be maintained constant, the lamp must be properly seasoned

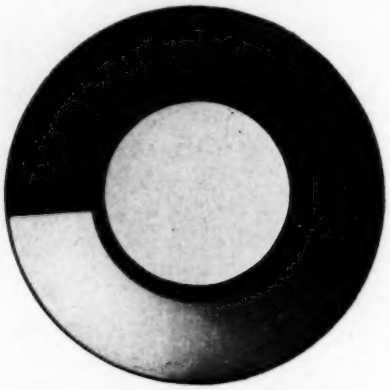

Fig. 6.—Face of photographic wedge.

and must be operated at a constant voltage. The seasoning of the lamp is accomplished by allowing it to burn from thirty-six to forty-eight hours while photometric tests of its candle power are made at intervals. When the initial rapid decrease in candle power has ceased, the lamp may be considered seasoned and may be safely used for a period of five hundred operating hours without any changes in candle power great enough to be of significance in testing the light sense. The voltage of the lamp terminals is controlled automatically by means of a voltage regulator designed by the Acme Apparatus Co. of Cambridge, Mass., especially for use with a 15 watt lamp. This attachment is one of the most important features contributing to the convenience and accuracy with which the instrument may be operated. The lamp may be connected to the ordinary 110 volt, 60 cycle alternating current [7] lighting circuit without the necessity of using a rheostat and a voltmeter to maintain constant voltage at the lamp terminals. With a variation in the line voltage of from 90 to 130, the voltage at the lamp remains at 110 within an error of not more than 1 per cent.

7. In the occasional instances in which alternating current of 60 cycles is not available, the voltage at the lamp must be maintained at 110 by means of a variable resistance and a voltmeter.

Variation of Brightness of Test Spot.—The mechanism by means of which the brightness of the test spot, i. e., of the luminous surface at A_2, is varied consists in a pair of photographic wedges, one of which is represented in figure 6. The wedges and the apparatus for making them have been described in previous publications, and consequently require only brief mention here. Each wedge consists of an annular strip of photographic film which has been so exposed to light that the density of the image decreases regularly in passing from the lightest portion around the ring to the darkest portion. When two wedges are mounted in such a way that the density varies in opposite directions, the resulting transmission is constant throughout the area of the wedge interposed in the path of the beam of light. The annular ring has a width of 2.5 cm.; consequently, the pair of wedges must be placed in the beam of light where its cross-section is less than this amount. If placed between the small aperture A_1, and the first lens l_1, it would be necessary to use wedges with an even gradation of density and without any flaws in order that the test surface at A_2 may be evenly illuminated. If, however, the wedges are placed in the collimated portion of the beam of light, such flaws are of no importance. In fact, it is not even necessary to use a pair of wedges; a single wedge may be used and an even illumination of the test surface at A_2 may still be obtained. This depends on the fact that the light reaching each point in the image is distributed evenly over the entire cross-section of the collimated beam of light. A similar method has already been used by Ferree and Rand in obtaining an evenly illuminated image. Their apparatus and the principles involved have been explained elsewhere in this paper. These authors use an adjustable diaphragm for varying the intensity. We have preferred to use photographic wedges, because they provide an easy means of varying the intensity continuously and gradually over a wide range. Since the logarithm of the transmission varies approximately with the distance along the wedge, the same amount of rotation of the pair of wedges everywhere produced approximately the same percentage change in the intensity of light. A pair of wedges was used in constructing this instrument, because at the time a single wedge having a sufficient density range was not available.

The density of the pair of annular wedges in the region through which the beam of light passes is varied by rotation of the wedges in opposite directions about an axis passing through the center of the rings perpendicular to the plane of the wedges. The wedges are caused to rotate by means of the handle shown in figure 1 at *H*. A shaft is connected at one end to this handle and at the other end to the center of the nearer wedge so that when the handle is rotated in a clockwise direction, the wedge also rotates in this direction. The two wedges are connected by means of fine chains which are wound around two drums and a series of pulleys in such a way that the rotation of the first wedge in a clockwise direction produces an equal rotation of the other in a counterclockwise direction and vice versa. The position of the wedges is indicated by means of a pointer and a scale graduated in degrees. These are shown in figure 5. Since the determinations of the light threshold are to be made in complete darkness, it is desirable that the operator be able to record automatically the position of the wedges corresponding to each determination of the light minimum. This may be done by inserting a pencil in the radial slot in the pointer (fig. 5) and making a mark on the paper scale beneath. The end of the pointer is covered with radium paint so that it may be located easily in the dark. The time at which a determination is made can be read from a clock having a luminous dial and can be recorded on the same scale. A fresh scale is inserted for each patient tested and serves as a record of the results of the test.

Calibration of the Scale.—The wedges were calibrated in position by making a series of readings of the brightness of the test spot corresponding to a series of scale readings at closely spaced intervals. A Macbeth illuminometer was used to make these determinations. In order to obtain intensities high enough to be measured with a satisfactory degree of precision, a 100 watt lamp was substituted for the 15 watt lamp while calibrating the wedges. In figure 7 is shown the calibration curve obtained. The brightness of the test spot when the scale reading is 335 degrees, i. e., when the thinnest portion of the wedges is in the path of the beam of light, is arbitrarily assigned a value of unity. The relative brightness

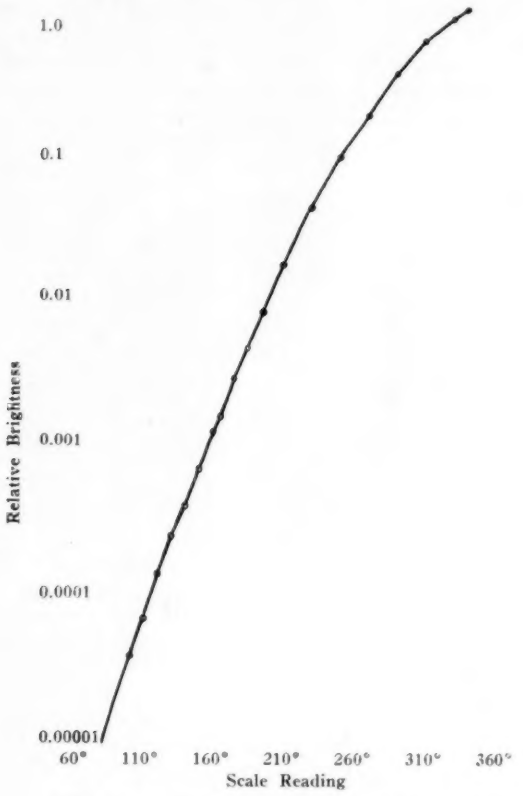

Fig. 7.—Calibration curve of pair of wedges.

is plotted on a logarithmic scale on the ordinate, and the corresponding scale reading is plotted on the abscissa. The resulting graph approximates a straight line, showing that the logarithm of the transmission is approximately proportional to the number of degrees through which the pair of wedges is rotated. In order to express the brightness of the test spot in absolute units of intensity, a single determination of the brightness of the test spot at a given scale reading must be made with the 15 watt lamp as the source of light, operated at the voltage that has been selected as standard. On the basis of this value any scale reading may be translated into absolute units of intensity. For example, if the brightness of the test spot with the scale at 335 degrees is 0.5 millilamberts or

TABLE 1.—*Method of Converting Scale Readings in Degrees into Intensity Units in Micromillilamberts*

Scale Reading	Intensity	Scale Reading	Intensity	Scale Reading	Intensity	Scale Reading	Intensity	Scale Reading	Intensity	Scale Reading	Intensity
		100	6.10	150	85.0	200	975	250	6,950	300	28,500
		101	6.48	151	90.0	201	1,030	251	7,200	301	29,100
		102	6.85	152	95.0	202	1,080	252	7,450	302	29,700
		103	7.23	153	100.0	203	1,130	253	7,700	303	30,300
		104	7.60	154	105.0	204	1,190	254	7,950	304	30,900
		105	7.98	155	110.0	205	1,240	255	8,200	305	31,500
		106	8.60	156	115.2	206	1,290	256	8,460	306	32,100
		107	9.23	157	120.4	207	1,340	257	8,720	307	32,700
		108	9.85	158	125.6	208	1,400	258	8,980	308	33,300
		109	10.5	159	131.0	209	1,450	259	9,240	309	33,900
		110	11.1	160	136	210	1,500	260	9,500	310	34,500
		111	11.8	161	146	211	1,570	261	9,780	311	35,100
		112	12.5	162	155	212	1,640	262	10,070	312	35,700
		113	13.3	163	165	213	1,710	263	10,350	313	36,300
		114	14.0	164	175	214	1,780	264	10,640	314	36 900
		115	14.7	165	185	215	1,850	265	10,920	315	37,500
		116	15.7	166	195	216	1,930	266	11,330	316	38,200
		117	16 6	167	205	217	2 010	267	11,730	317	38,900
		118	17.6	168	215	218	2,090	268	12,140	318	39,600
		119	18.5	169	226	219	2,170	269	12,540	319	40,300
		120	19.5	170	236	220	2,250	270	12 950	320	41,000
		121	20.6	171	250	221	2,350	271	13,360	321	41,520
		122	21.7	172	263	222	2,450	272	13,770	322	42,040
		123	22.8	173	277	223	2,560	273	14,180	323	42,560
		124	23.9	174	290	224	2,660	274	14,590	324	43,080
75	1.25	125	25.0	175	304	225	2,760	275	15,000	325	43,600
76	1.35	126	26.5	176	318	226	2,890	276	15,450	326	44,280
77	1.45	127	28.0	177	331	227	3,020	277	15,900	327	44,960
78	1.55	128	29.5	178	345	228	3,144	278	16,350	328	45,640
79	1.65	129	31.0	179	358	229	3,272	279	16,800	329	46 320
80	1.75	130	32.5	180	372	230	3,400	280	17,250	330	47,000
81	1.90	131	33.8	181	398	231	3,550	281	17,750	331	47,600
82	2.05	132	35.0	182	323	232	3,700	282	18,250	332	48 200
83	2.20	133	36.3	183	349	233	3,850	283	18.750	333	48,800
84	2.35	134	37.5	184	374	234	4,000	284	19,250	334	49,400
85	2.50	135	38.8	185	500	235	4,150	285	19,750	335	50,000
86	2.70	136	41.4	186	524	236	4,320	286	20,400		
87	2.90	137	44.1	187	547	237	4,490	287	21,000		
88	3.10	138	46.7	188	571	238	4,660	288	21,600		
89	3.30	139	49.4	189	594	239	4,830	289	22,300		
90	3.50	140	52.0	190	618	240	5,000	290	22 900		
91	3.72	141	54.6	191	649	241	5,165	291	23,400		
92	3.93	142	57.2	192	681	242	5,330	292	23 900		
93	4.15	143	59.7	193	712	243	5,495	293	24,500		
94	4.36	144	62.3	194	744	244	5,660	294	25,000		
95	4.58	145	64.9	195	775	245	5,825	295	25,500		
96	4.88	146	68.9	196	815	246	6,050	296	26,100		
97	5.19	147	72.9	197	855	247	6,275	297	26,700		
98	5.49	148	76.9	198	895	248	6,500	298	27,300		
99	5.80	149	81.0	199	935	249	6,725	299	27,900		

TABLE 2.—*Factors by Which Intensities Read from Table 2 Are to Be Multiplied in Order to Correct for Size of Pupil*

Diameter of Pupil in Millimeters	Correction Factor
1.0	0.04
1.5	0.09
2.0	0.16
2.5	0.25
3.0	0.36
3.5	0.49
4.0	0.64
4.5	0.81
5.0	1.00
5.5	1.21
6.0	1.44
6.5	1.69
7.0	1.96
7.5	2.25
8.0	2.56
8.5	2.89
9.0	3.24

50,000 micromillilamberts, the brightness corresponding to any other scale reading is obtained by reading off the proper value of relative transmission from the graph and multiplying it by 50,000. By this means table 1 has been constructed, for every point on the scale the corresponding brightness in micromillilamberts being given. If this table is to give correct readings when different lamps are inserted in the instrument, care must be taken that only those lamps are used that have the same candle power as the original lamp with which the value of 50,000 micromillilamberts was obtained. If lamps differing appreciably in candle power are used, a correction factor must be applied to the data obtained from the table. As may be seen from table 1, between the limits of 335 and 75 degrees on the scale, the brightness of the test spot varies from 50,000 to 1.25 micromillilamberts. The scale was not calibrated below 75 degrees, because beyond this point on the scale the 100 watt lamp did not give intensities great enough to be measured with precision, and because from our previous investigations a range of from 50,000 to 1.25 was known to be more than adequate for all possible cases.

General Considerations.—The instrument described should prove feasible for use by the practicing ophthalmologist. It is small, measuring 32½ by 9½ by 7½ inches and weighs only 23 pounds. It is furnished with a handle by means of which it may be carried as easily as an ordinary suitcase. A metal shutter

TABLE 3.—*Data and Calculation in Typical Case*

2 mm. pupil

Time, Minutes	Scale Reading	Intensity, Micromillilamberts	Intensity Corrected for Size of Pupil, Micromillilamberts
1½	193	712	114
14½	137	44.1	7.06
18¾	128	29.5	4.82
26½	120	19.5	3.12
33	106	18	2.88

is provided for the glass test surface in order that it may be protected from breakage when the instrument is carried about. The compartment containing the source of light is so constructed that no stray light escapes; consequently, the apparatus does not need to be enclosed, but may simply be placed on a table in the dark room at the proper distance in front of the patient.

PROCEDURE USED IN MAKING DETERMINATIONS

The procedure used by us in making a determination of the course of dark adaptation has been described in a previous article.[8] It may be of value, however, to consider here possible simplifications which may be made in using the instrument for routine diagnosis instead of for research purposes. The advantages and disadvantages of making these simplifications will be pointed out. At the end of this discussion, a routine procedure of the simplest possible sort will be outlined briefly.

Pupil Fixation.—Our method of correcting for the variable effect of the results of different sizes of pupil consists in immobilizing the pupil with pilocarpine, measuring its diameter, and applying to the results of the determination

8. Footnote 2 (first reference).

of the light threshold a correction factor based on the area of the pupil. The pupillary diameter is measured by means of the Zeiss slit-lamp and corneal microscope. The pupil, illuminated by the intense beam of light of the slit-lamp, is viewed under a magnification of 13, and its diameter measured by means of the scale in the ocular micrometer. Although the involuntary eye movements of the patient, magnified of course in the same ratio, are a source of error and the scale divisions are seen with difficulty because the scale itself is not well illuminated, determinations of the pupil diameter may in most cases be made to within one scale division. For a pupillary diameter of approximately 1.5 mm., an error of measurement of one scale division corresponds to an error of 11 per cent in the area. Since the pupils fixed with pilocarpine in this way are rarely smaller than 1.5 mm., the error of measurement is in general not greater than 11 per cent. If a slit-lamp is not available, the measurement may be made by means of any of the various types of pupillometers. With an ordinary keratometer, it is possible to measure the pupillary diameter to within 0.25 mm., and thus to compute the proper correction factor with an error not greater than about 33 per cent, or for pupils measuring 1.5 mm. or more. The measurement of the pupillary diameter may be made both before and after the threshold determinations in order to be sure that there has been no appreciable change in the size of the pupil during the course of the test. Our experience has shown, however, that if the pupil does not react to light after thirty minutes have elapsed following the instillation of 3 drops of a 1 per cent solution of pilocarpine, determinations of its diameter made before and after the tests in the dark room agree within the error of the measurements.

Preexposure.—The course of dark adaptation will obviously be influenced by the level of illumination to which the eye has been exposed immediately before the adaptation to darkness begins. In order to eliminate the variability in the results due to this factor, the following procedure was adopted:

The observer is seated facing a white screen 27 inches distant, which fills practically the entire field of view. The screen is illuminated to a brightness of approximately 21 millilamberts (19.5 apparent foot candles) by means of two 100 watt mazda lamps mounted on adjustable floor stands and placed out of the patient's line of vision at a distance of approximately 4 feet from the screen. The patient is allowed to look at this screen for a period of ten minutes or more in order that a constant level of light adaptation may be reached. At the end of ten minutes the lights are turned out, the exact time at which this is done being recorded in order to determine accurately the instant at which the process of dark adaptation commences. The screen having been removed from its position in front of the patient, the test spot is exposed, and the determinations of the light threshold may be made at the desired intervals during the course of dark adaptation.

Although the amount of light reaching the patient's retina from this screen will vary with the size of the pupil, we have found that the course of dark adaptation is affected by this only in the early stages and by an amount which may be considered negligible. Slight variations in the brightness of the preexposure screen may also be considered of minor importance, although a decrease in brightness below the standard level produces a greater effect on the results than an equal increase. If, therefore, it is not possible to obtain an illumination of the preexposure screen approximately equal to the chosen standard, it is better to use a higher brightness than a lower. If no other means are available, it may be possible to obtain a preexposure screen of the proper brightness by substituting a plain white card for the acuity chart and using the source of light ordinarily employed in illuminating this chart. When possible, the preexposure screen

should be set up in the dark room in which the determinations of the light minimum are to be made. In this way only can the preexposure immediately preceding the beginning of dark adaptation be controlled.

Threshold Determinations.—Although an exact control of fixation is probably not necessary or feasible for patients seen in the clinic, the procedure followed in measuring the light minimum should be such as to insure that for a given patient approximately the same part of the retina is tested at each determination. If a series of short exposures of the test spot at different intensities are made and the patient is asked to indicate whether or not he sees it, he is likely to be looking in quite a different direction each time, and as a result may see the spot occasionally with the more sensitive periphery and occasionally with other less sensitive parts of the retina.

In order to prevent too much variability in result due to varying conditions of fixation, the following procedure should be used in making each determination of the light threshold: The approximate threshold is first found by darkening the spot rapidly until it disappears and then, while the patient continues to look in the direction in which it was last seen, increasing its brightness rapidly until it appears again. This procedure is repeated a second time with a more gradual variation in the brightness of the spot, and the intensity at which the spot is first seen when it is increased gradually in brightness is recorded as the threshold. Investigations as yet unfinished suggest that in fixating a dim object when the eye is partially or completely dark-adapted the tendency is not to turn the eye so that the image falls on the fovea, but rather to take a slightly eccentric fixation so that the image falls on some other more sensitive part of the retina, which may be called the scotopic fovea. Whether or not this tentative conclusion will be confirmed by more extended investigation, we cannot say. Actual experience has shown, however, that whatever may be the explanation, the procedure just outlined for determining the threshold gives more consistent and reproducible results than are obtained when a series of short exposures of the test spot at different brightnesses are given.

Method of Computing Data.—A typical series of determinations for a normal observer with a pupil 2 mm. in diameter is shown in table 3. Column 1 gives the time at which each measurement of the slight threshold was made and column 2, the corresponding scale reading. By means of table 1, these readings are converted into the micromillilambert values given in column 3. The next step consists in expressing the results in terms of the number of micromillilamberts which would have been required for the threshold if the patient's pupillary diameter had been 5 instead of 2 mm., the former having been chosen arbitrarily as the standard value. These, the final data given in column 4, are obtained from column 3 by multiplying each number in column 3 by 0.16, the correction factor for a 2 mm. pupil. Table 2 gives the correction factors for each diameter of pupil. The final data are shown graphically in figure 8, in which time is plotted along the abscissa and the corresponding brightness is plotted according to a logarithmic scale along the ordinate.

Evaluation of Data.—The graph shown in figure 8 also contains a curve representing the upper limit of normal variation. This curve and the details of its determination have been discussed in a previous paper.[2] The necessary data were obtained by testing a number of normal subjects with the older and more elaborate form of apparatus previously described. It is probable that the curves for a given person tested by both machines would not differ greatly from one another. Since, however, the color quality of the light sources and the shapes of the test spot are different, an investigation of this question must be made. If

any consistent difference of an appreciable amount is found in the data obtained with the two instruments, it will be necessary to make a similar determination of the limits of normal variation with the present apparatus. A comparison of the results obtained with the two machines is now in progress.

Brief Summary of Essential Steps in Simplified Procedure.—The following statement summarizes what we believe to be the simplest possible procedure which can safely be used in making a test of the course of dark adaptation, without neglecting considerations essential to the value of the test in diagnosis:

Three drops of 1 per cent pilocarpine are instilled at three minute intervals. Fixation of the pupils may be considered complete at the end of thirty minutes.

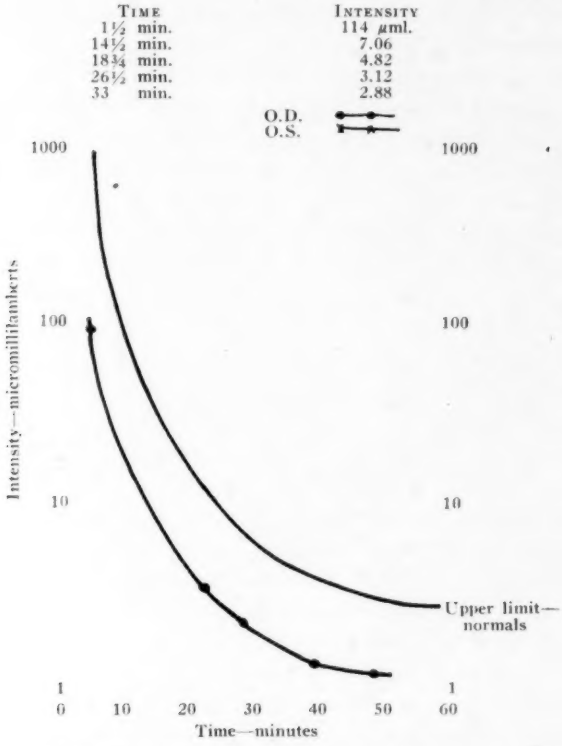

Fig. 8.—Dark adaptation curve of a normal eye in W. S. A., a man, aged 54 years, on May 23, 1929. A diagnosis of normal light sense in the right eye was made. The solid line indicates the left eye and the line broken by black circles, the right eye.

The last ten minutes of this period is employed in the light adaptation of the eyes. During this time, the patient is seated facing a white screen illuminated by an intensity of light not less than that given by a 100 watt lamp placed 3 feet away and behind the patient. In case such an arrangement is not available, the patient may sit 2 feet from the illuminated test chart or at a window where the skyline is visible and where there is good daylight. At the end of the preexposure period, the patient is transferred to the dark room, and the exact time at which dark adaptation begins is recorded. The patient is seated facing the apparatus

at a distance of 1.33 meters from the test spot. As soon as possible, determinations of the light threshold are made, first on one eye and then on the other, according to the directions previously given. When one eye is being tested, the other is kept covered by the patient's hand. In order to avoid confusion, the readings for the two eyes may be recorded with differently colored pencils. Three subsequent readings are made at ten minute intervals, requiring the patient to remain in the dark room a little more than thirty minutes. The operator need stay in the dark room only when readings are being taken, but he must take care that the patient's eyes are closed and covered with the hands when he is entering or leaving the room. At the end of the test, the diameters of the pupils are measured. The results are then calculated according to the directions given elsewhere in the paper, and the final data plotted on a chart on which the normal curve is represented.

SUMMARY

The essential features of an instrument to be used in measuring the changes in the light threshold during dark adaptation are outlined. The qualifications of several of the more important adaptometers already in use are considered in relation to these requirements. A simple instrument is described which fulfils these requirements and is suitable for use in a physician's office. The procedure to be followed in making a determination of the course of dark adaptation is outlined.

THE TRANSVERSE SINUS AND ITS RELATION TO CHOKED DISK *

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Since Turk in 1853 called attention to retinal congestion and hemorrhages in all tumors of the brain, and von Graefe in 1860 described "choked disk" and attributed the condition to pressure, either direct or indirect, against the cavernous sinuses, both opththalmologists and neurologists have tried to explain, through various theories and many experiments, the actual mechanism whereby the disk becomes congested, edematous and, finally, choked.

In a previous paper [1] attention was called to the various theories and a return to the mechanical theory was suggested as the probable, or at least a reasonable, solution to the problem. In that paper no effort was made to explain the absence of choked disk in certain cases of tumor of the brain in which normally one would expect to find a marked choking, for instance, in tumors of the cerebellopontile angle, but I believe that a reasonable explanation can be found in the anomalies of the venous sinuses. During the past year, I have observed three cases which have a definite and distinct bearing on this subject, all of them having to deal with faulty development in the venous dural sinuses. These cases will be described in detail at the end of this paper, but for present purposes a brief outline is essential.

The first case was that of a woman, aged 76, seen at autopsy, who had for twenty-six years been having periodic convulsive seizures, and who after seven days of unconsciousness died in a convulsive seizure. The autopsy showed a tumor the size of a walnut situated at the cerebellopontile angle on the left side, but the transverse and sigmoid sinuses on that side were of rudimentary character (fig. 1). Practically no blood was found in the sinus itself, although the inferior petrosal sinus drained into the jugular bulb and was almost of normal size. She came of a Christian Scientist family, and it was difficult to get a history, but sufficient was obtained to note that she had had dizziness over a long period and finally deafness of the left ear.

The second case was that of a girl, aged 21, who gave a history of intense pain with nausea and vomiting, double choked disk and double Babinski reflex of two weeks' duration. At autopsy a tumor of the brain was found in the lateral and inferior border of the occipital

* Submitted for publication, April 20, 1929.

* Read before the Section on Ophthalmology at the Eightieth Annual Session of the American Medical Association, Portland, Ore., July 11, 1929.

1. Swift, G. W.: Choked Disk in Intracranial Lesions: The Mechanical Factors in its Causation, Northwest Med. **26**:579 (Dec.) 1927.

lobe on the right side. This tumor rested on the right transverse sinus at its juncture with the sigmoid portion, while the left transverse and sigmoid sinuses were rudimentary in character (fig. 2).

In other words, in case 1, practically the entire venous blood from the brain was carried out of the cranial cavity by the right transverse sinus, while the tumor was situated against the left sigmoid. In case 2, the entire venous blood from the brain was carried out by the right transverse sinus and the tumor was situated immediately above this sinus and compressed the sinus walls. The brain in case 1, in which the pressure was against an obliterated sinus, showed deep and wide sulci, with lakes of cerebrospinal fluid between the convolutions, as found in typical idiopathic epilepsy; in the second case, the convolutions were all wide and flat, the sulci were obliterated, and there was an

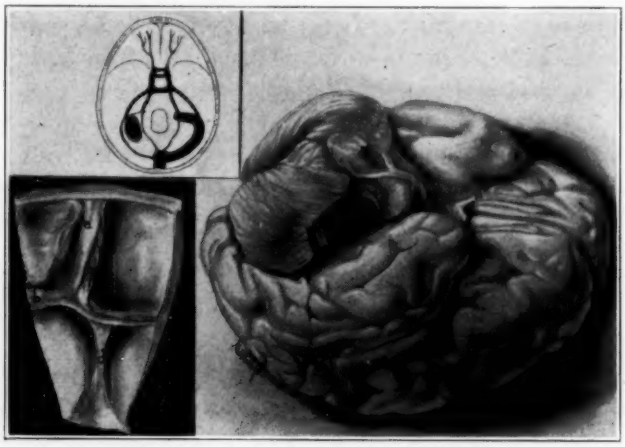

Fig. 1 (case 1).—Tumor of the left cerebellopontile angle with cystic degeneration in a case of idiopathic epilepsy. Convolutions of the brain narrowed and cortex atrophic. Superior longitudinal sinus (*a*) empties into right transverse sinus (*b*). Left transverse sinus (*c*) rudimentary in character. Choked disk did not occur because the tumor did not press on the return venous blood in the right transverse dural sinus.

intense edema of the brain itself, with moderate dilatation of the ventricles. The skull in each instance showed a definite wide groove for the large transverse sinus and a narrow, almost ridgelike appearance for the left sinus, which had not developed. The eyes of the first patient, the epileptic woman, did not present a choked disk at any time, nor did the tumor cause a damming up of the venous return from the cranial cavity; in all likelihood it played a very insignificant part, if any, in the death of this patient. In the second case, when the tumor had reached the point at which there was sufficient pressure on the transverse sinus at its junction with the sigmoid, a stasis of the entire venous blood

within the cranial cavity resulted and the cerebrospinal fluid could no longer escape. The double choked disk was due entirely to a shutting off of the return blood from the orbits, by both direct and indirect pressure.

Another observation which was made in both these cases was that the jugular foramen on each side of the rudimentary sinus (left) was extremely small in diameter, in comparison with the extra wide jugular foramen on the opposite (right) side. Obviously the returned blood from the cavernous sinus on the side of the rudimentary sinus had long since been short-circuited to the opposite side and was cared for by the superior and inferior petrosal sinuses of the opposite side, with

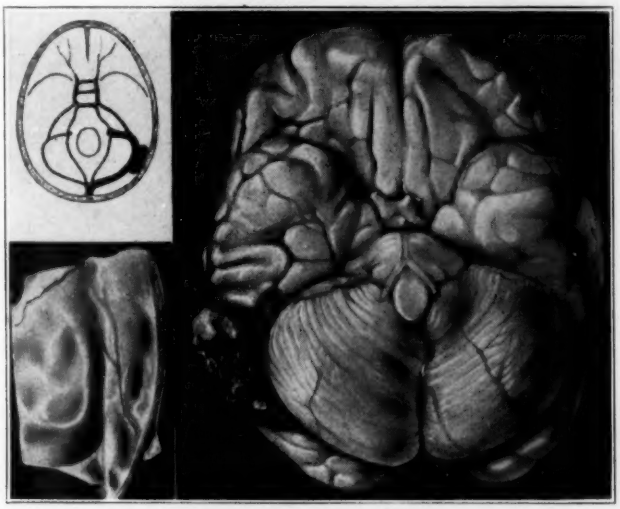

Fig. 2 (case 2).—Tumor of the right occipital lobe, lateral inferior margin, attached to the right transverse sinus where the superior bone (*a*) marks the site of the right transverse sinus where the superior longitudinal sinus empties; *b*, a small groove for the rudimentary left transverse sinus. Illness of short duration with a fulminating choked disk because the tumor presses on the right transverse sinus, which carries practically all of the cerebral blood from the superior longitudinal sinus. Indirect pressure causes the choked disk, since the right inferior and superior petrosals are not blocked off by direct pressure.

possibly a small amount by way of the inferior petrosal sinus of the same side.

To summarize these two cases, one might say that in two women, one 76 and the other 21, the right transverse sinus was fully developed while the left was only a rudimentary structure. In the first case the tumor occurred on the left or undeveloped side; in the other, on the right or sinus functioning side. In the first case no symptoms either of increased intracranial pressure or of choked disk developed, while in

the other there was a sudden fulminating chain of symptoms—nausea, vomiting, headache, choked disk, mental confusion—and finally death, all during a few weeks' time.

The third case was that of a new-born child who died in status epilepticus on the morning of the third day after birth. Autopsy revealed extensive hemorrhages in the subdural spaces and particularly in the posterior fossa. A careful examination at the time showed that

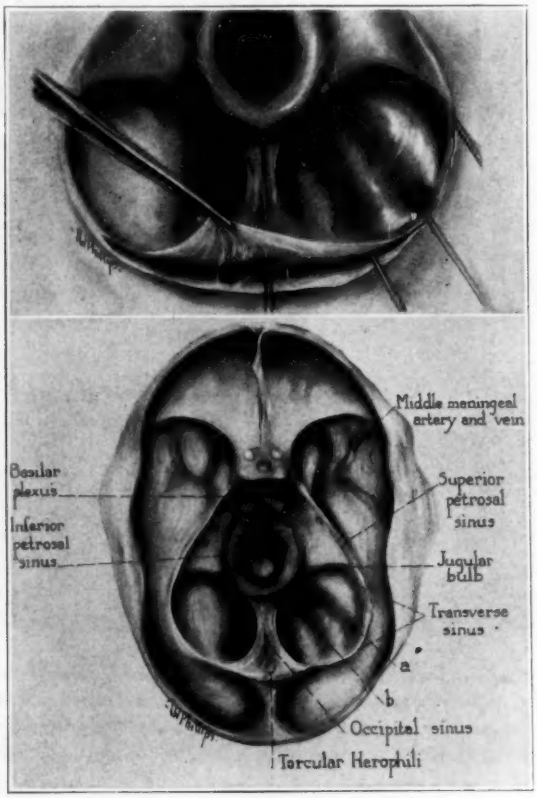

Fig. 3 (case 3).—Development of atypical dural sinuses with occlusion of the jugular foramina; the child lived only three days: Above, basilar sinuses in a new-born child; there is a faulty development of the jugular bulbs and several anomalous dural sinuses are present without well formed walls. Below, detail of the transverse sinuses, showing trabeculae which are remnants of the primary plexuses.

in this particular child the trabeculae which usually disappear in the transverse sinus were still present, the rudiments of embryologic development (fig. 3); that the jugular foramina were both rudimentary in character and small in size, and that the intracranial venous pressure

was great enough to spread the newly formed venous sinuses and widely separate the two layers of dura. Large lakes of blood replaced the occipital sinuses, while on the left side two newly formed sinuses coursed down to the site of the jugular foramen. This peculiar faulty embryologic development may account for many birth hemorrhages. The eyes in this case were examined prior to death and hemorrhagic retinitis was present, as in many new-born children with intracranial hemorrhages.

EMBRYOLOGY

An important factor which one must first understand in order to appreciate what takes place in certain intracranial conditions is the embryologic development of the dural sinuses. Streeter [2] made a comprehensive study of these sinuses. He showed conclusively that in the

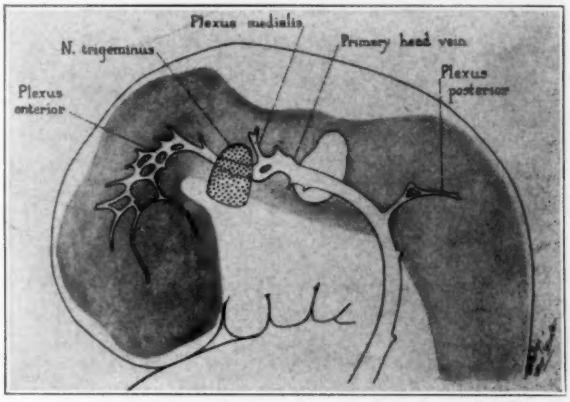

Fig. 4 (after Streeter).—Beginning of the dural sinuses: the anterior primary plexus partially formed, the middle and posterior just beginning.

embryologic development of the human embryo certain primary plexuses are eventually replaced by the development of the dural (fig. 4) sinuses; these primary plexuses are divided into an anterior, middle and posterior portion, draining through larger veins to join the future jugular vein (fig. 5). As the embryo develops, the larger portion of the anterior plexus anastomoses freely with the middle plexus and drains into the posterior plexus by a newly formed channel dorsal to the primary head vein (fig. 6). This new channel eventually becomes the lateral sinus, while what remains of the original anterior plexus eventually becomes the ophthalmic vein and the cavernous sinus. The primary head vein, which at first drains the anterior and middle primary plexuses, after the

2. Streeter, G. L.: The Development of the Venous Sinuses of the Dura Mater in the Human Embryo, Am. J. Anat., 1915, vol. 18.

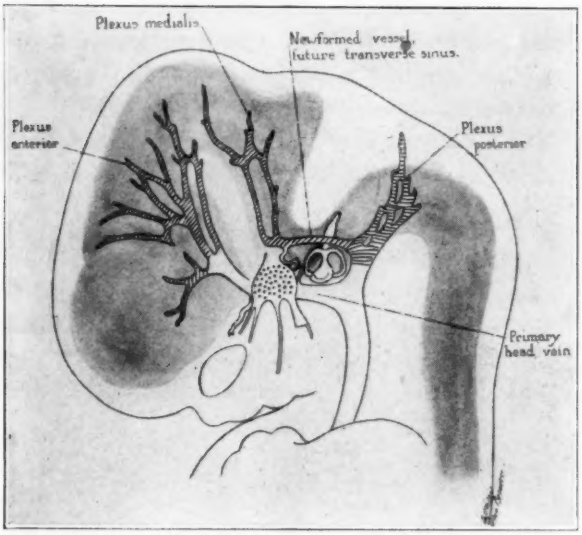

Fig. 5 (after Streeter).—Anterior, middle and posterior primary plexuses rapidly developing; shaded portion shows the beginning of the permanent dural sinuses.

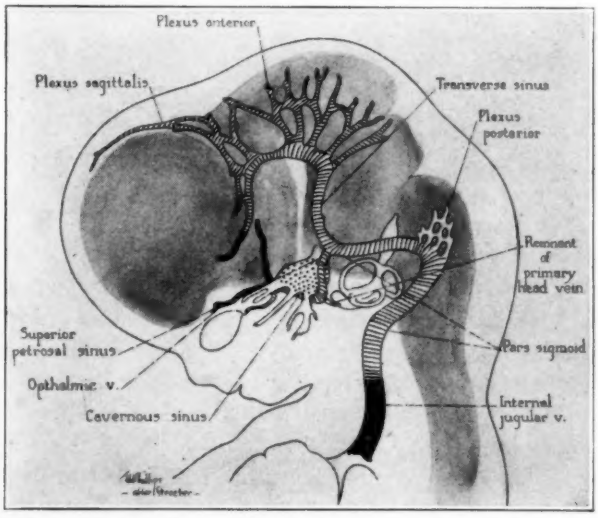

Fig. 6 (modified from Streeter).—Embryo 21 mm. long: From the anterior plexus develops the future cavernous sinus; the jugular bulb is already formed; the primary head vein has disappeared except for a remnant just behind the cavernous sinus; the future superior petrosal sinus appears at this time; the brain begins to enlarge and displace the anterior plexus backward.

formation of the dorsally placed new vein, gradually becomes smaller and smaller, and its anterior portion becomes the cavernous sinus (fig. 7). This sinus originally receives as tributaries the ophthalmic veins and a large cerebral vein which drains the lateral wall of the diencephalon. Later this vein disappears, as do many other cerebral veins which first drain into the dura and later drain through the cerebral venous system (fig. 6).

The original trunk of the middle dural plexus, which had almost disappeared with the development of the dorsal anastomosing trunk between the anterior, middle and posterior plexuses (figs. 4 and 6), now assumes a larger size and becomes the future superior petrosal sinus, while the primary head vein, which had been the large vein drain-

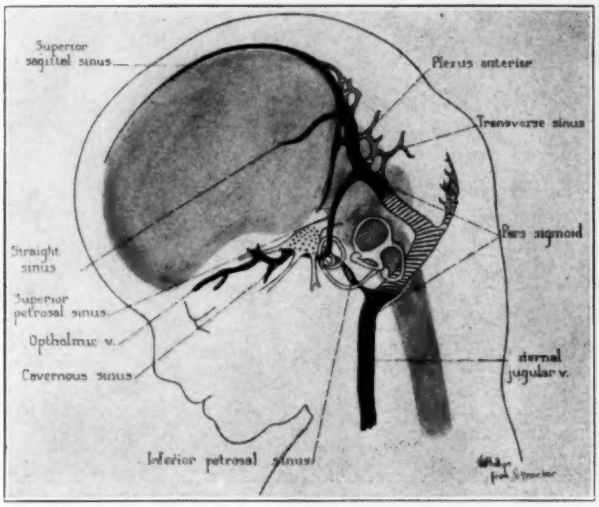

Fig. 7 (modified from Streeter).—Embryo 50 mm. long: The straight sinus and the inferior petrosal sinus appear for the first time; the superior sagittal is now well developed.

ing both the superior and the middle plexuses, becomes a rudimentary structure in the human being. It sometimes persists, and a foramen known as the "foramen jugulare spurium" of Luschka is sometimes found in adult skulls. In animals, however, this vein connects with the external jugular vein and offers an escape of the blood from the cavernous sinuses which is not present in the human being. The cavernous sinus now drains upward over the semilunar ganglion through what was the original middle dural vein and is now known as the superior petrosal sinus. The new channel which has formed for the drainage of what remains of the anterior dural plexus and of the entire middle and posterior dural plexus empties into the jugular vein and becomes

the transverse and sigmoid sinuses. This development of the sinuses occurs approximately in an 18 mm. embryo (fig. 8).

As the cerebrum and cerebellum begin to grow, a readjustment of the venous channels must take place. The first changes occur approximately between 18 and 21 mm., involving the anterior dural plexus (fig. 9). This plexus coalesces and practically absorbs the middle plexus. This is the beginning in the midline of the future superior sagittal sinus and, as the brain gradually enlarges, this new sagittal sinus is forced gradually backward and, as it does so, pushes the newformed lateral sinus downward and outward, rotating it to the horizontal plane (fig. 9).

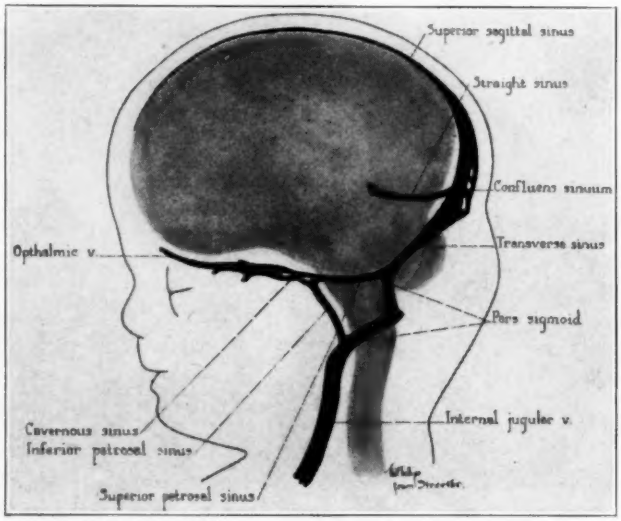

Fig. 8 (modified from Streeter).—Embryo 80 mm. long; all sinuses fully developed.

The development of the labyrinth from now on adds a complicating factor. This development consists of a lateral displacement of the lateral sinus and the development of the sigmoid sinus. When the embryo is 50 mm. long, the venous channels are approximately the same as found at birth (fig. 8).

FURTHER DEVELOPMENT OF THE DURA AND SINUSES

Streeter [2] said:

On examining the dura in embryos 50 mm. long, it will be seen that for the greater part it closely invests the interior of the developing cranium and is relatively poor in blood vessels. This is true especially in those portions where the cartilaginous and bony cranium is more advanced in its differentiation, as in the base of the skull and in the frontal, temporal and lower occipital regions. In

other regions the dura projects within the cranial cavity, being separated from the future bony skull by a layer of areolar tissue, in the meshes of which are found the large blood channels and their tributaries. The largest area of this kind is found situated over the midbrain and extending from the caudal margin of the cerebral hemispheres to the cerebellum. This area extends laterally down to the base of the skull, narrowing as it does so. It constitutes what is known later as the "tentorium cerebelli" and in it is included the greater part of the dural venous system. A basal extension of the tentorium widens out in the region of the semilunar ganglion and in its meshes is formed the cavernous sinus. A thinner area of the same tissue extends caudalward from the cavernous sinus, medial to the otic capsule, to join the jugular region. *The slender plexus of veins extending through this constitutes the inferior petrosal sinus.* Along all the sinuses we find this same areolar meshwork. It is not to be confused with the developing arachnoid tissue, from which it is everywhere separated by the dura. Blood vessels supplying and draining the brain are also found in the arachnoid at this time; they are quite numerous in some regions, such as the region of the sylvian fissure and along the more ventral parts of the midbrain and hindbrain. These cerebral vessels are everywhere separated and distinct from the dural

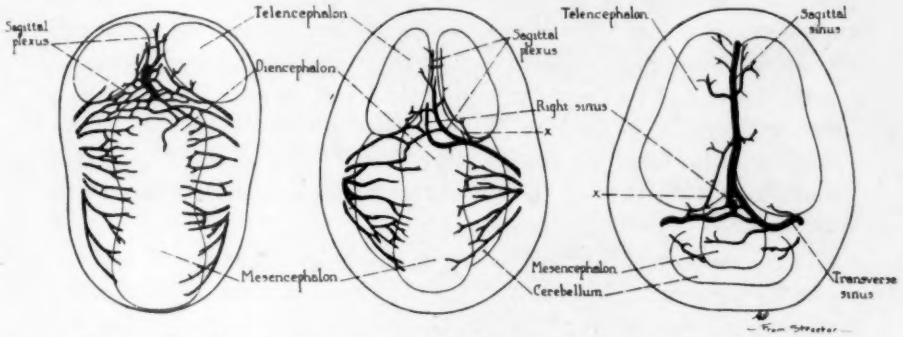

Fig. 9 (modified from Streeter).—Formation of the superior sagittal sinus, showing the channel selection with the possibilities of anomalies.

blood channels, with the exception of the few points where they empty into the big dural channels, as occurs in the adult. The connection between the dural system and the cerebral system is no longer by a multiple anastomosis of small vessels but, instead, by isolated larger veins.

In the development of the transverse sinus, the first part to develop is the terminal or jugular portion (fig. 6). This extends upward to the point of entry of the superior petrosal sinus and is known as the sigmoid portion. The transverse sinus proper is developed later and has a large capillary meshwork along its dorsal margin which is the remains of the anterior dural plexus. Along the anterior margin of this plexus will form the main channel into which the inferior cerebral vein empties (fig. 9). This channel has migrated backward during the growth of the hemispheres until it finally assumes a horizontal course and has now reached approximately its adult development, and the variations found at this point in adults must be considered as variations

in channel selections in this meshwork. In any structure that develops as the result of the coalescence of a series of channels, the channel selection plays an important part in the ultimate development of that particular structure. This undoubtedly accounts for the peculiar anomalies which are found in the development of these sinuses. The development of the bony labyrinth probably plays an important part both in the passive migration causing flexion and in the spontaneous migration, where new channels are developed.

SUMMARY OF THE EMBRYOLOGIC DEVELOPMENT

From the original work of Streeter [2] and reports by other observers (Streit, Labbe, Malacre, Knott, Sperino, Hallett, Meckle, Dumont, Heine and Beyer [3]) it must be concluded that embryologically the constant portions of the venous dural sinuses are the ophthalmic veins, cavernous sinuses, sigmoid sinuses and jugular bulbs. The variable portions are due to the migration, passive and spontaneous, and the coalescence of a number of smaller channels into larger ones ultimately the dural sinuses. As both the migration and the channel selection may be influenced by the enlarging cerebral hemispheres, the coincident bone development of the base (basilar occipital centers) and the rather distorting otitic labyrinth development, it follows that one may expect variations in that part of the venous dural sinuses more likely to be thus involved.

That such is true is abundantly proved in the statistics of Streeter as to the asymmetry of the superior longitudinal sinus with the two transverse sinuses, 89 per cent of all cases showing more development on the right side. Reports of many observers quoted by Braun and others show the absence of one or the other transverse sinus, double right or left sinuses replacing a single transverse sinus, branched longitudinal sinuses, large occipital sinuses draining into the vertebral veins or across the fossa to the jugular bulb, and many other minor variations (figs. 10-15). Practically all variations have, however, involved only the superior longitudinal, transverse and sigmoid sinuses. The variations of the sigmoid are entirely due to the size of the transverse sinus and the body development of the mastoid portion of the temporal bone. The size, shape and position of the jugular bulb depend on the development of the mastoid bone. Braun,[3] Prentiss, Rudolph, Streit, Rohrbach and practically all other observers agree on the constant position of the two petrosal sinuses and on the arrangement of a constant cross connection between the two cavernous sinuses, so that blood from the right can easily flow into the left and vice versa.

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3. Braun, Alfred: Sinus Thrombophlebitis: Inflammatory Diseases of the Venous Sinuses of the Dura Mater, 1928.

These are important factors to be considered in the relationship of pressure and stasis in the development of choked disk.

It would then follow that under normal conditions of life, granting that with the fetal development sufficient outlet capacity had developed for the venous return from the cranial cavity, the establishment of the

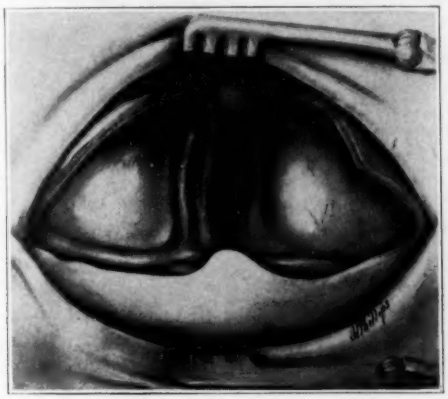

Fig. 10.—Marked enlargement of inferior occipital sinuses; almost rudimentary left transverse sinus.

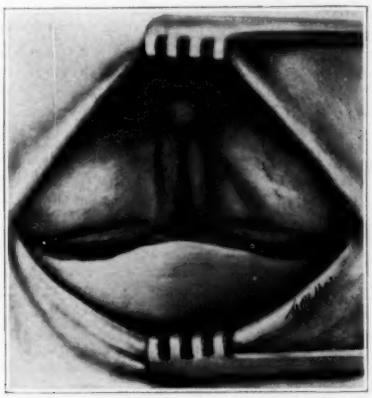

Fig. 11.—Right and left transverse sinuses small; occipital sinuses both very large.

infant circulation at birth would easily follow. Should sufficient outlet, i. e., jugular vein capacity, not be developed, the arterial inflow would soon overcome the venous outflow, with the result illustrated by case 3. It is only when an abnormal change takes place within the cranial cavity, such as a new growth encroaching on the normal spaces, a large hemorrhage or edema of a general character, that one finds

occasion for disturbances in the circulation within the sinuses, even though an abnormally developed system of dural sinuses is present. When such occurs, the resultant effects on the circulation within the

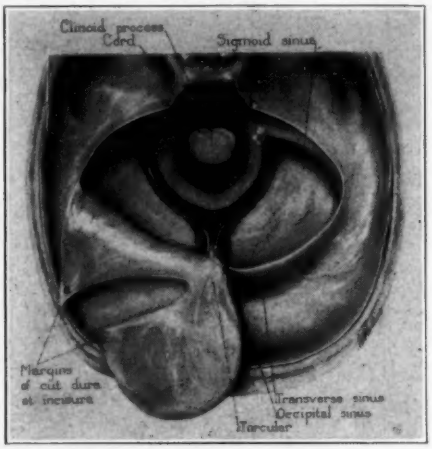

Fig. 12.—Occipital and transverse sinuses very large; jugular foramina highly developed; in such a case choked disk is practically impossible, no matter where the tumor is situated. This patient had an enormous intracranial hemorrhage with no symptoms for seven days after injury; then there was sudden coma and death (Coroner's case).

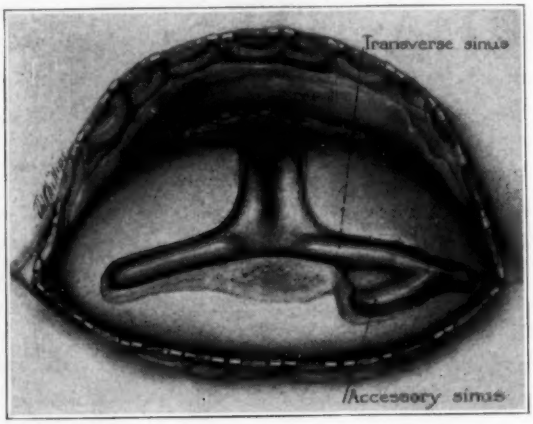

Fig. 13.—Anomalous or accessory transverse sinus due to faulty channel selection in the embryologic development.

sinuses and the effect on the return venous orbital blood become apparent. In the normal circulatory arrangement, the effect of a general increased intracranial pressure is a gradual development of congestion of the veins of the retina and disk. As this develops to a

greater and greater degree, however, edema first of the disk (Parsons) and later of the retina develops (fig. 16). A rapid development of pressure within the cranial cavity causes a quite different appearance of the disk from a slowly progressing pressure—acute fulminating conditions, such as arteriovenous aneurysms of traumatic character, in the cavernous sinus, suffocation in mines, dynamite explosions, edema of

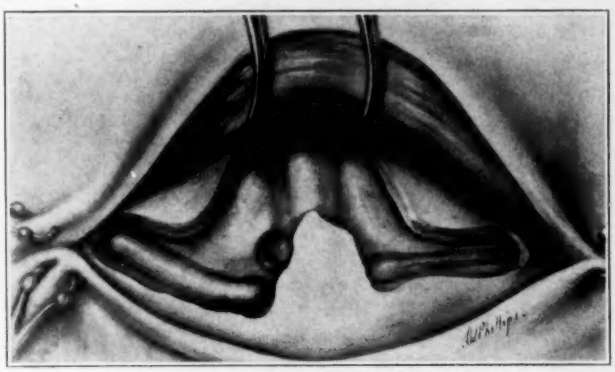

Fig. 14.—Aneurysmal dilatation of right transverse sinus just external to the torcular.

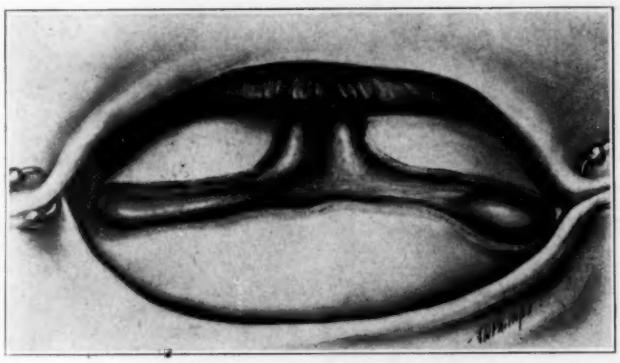

Fig. 15.—Knoblike dilatation of both transverse sinuses at junction with the sigmoid.

the brain in extreme thrombophlebitis (fig. 17), intraventricular hemorrhages, tumor or syphilis. The veins literally rupture in such cases. By way of contrast, the gradual edema occurring in a case of hemorrhage with slow edema and slowly enlarging tumors of the thalamic region is of interest (fig. 18).

The direct pressure against a sinus draining the right or the left eye can be located only in the region of (1) the cavernous sinus (fig.

19) and (2) at the jugular bulb (fig. 20). This pressure may occur in aneurysms of the cavernous sinus, lateral sinus thrombosis in mastoid disease, tumors or abscesses of the temporosphenoidal lobes (anterior tip), and cerebellar tumors and cysts.

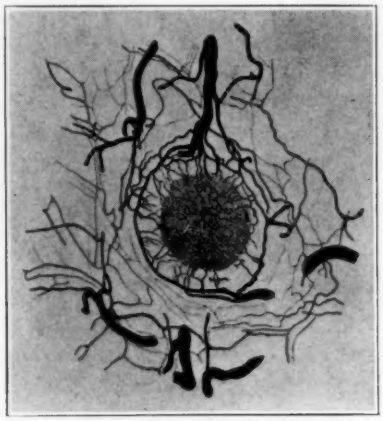

Fig. 16 (after Parsons).—Dilatation of the retinal veins and capillaries of the optic nerve, the free anastomosis and the development of edema in the disk itself.

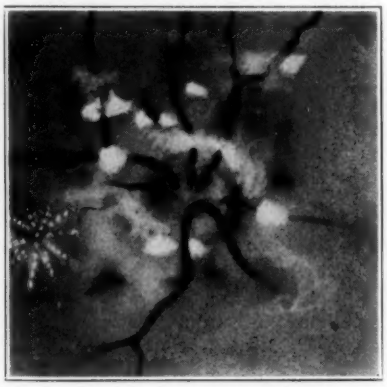

Fig. 17.—Massive exudate covering tortuous vessels with spontaneous rupture appearing frequently; disks are entirely obscured. The pressure was due to an edema of the brain, the result of a thrombophlebitis with increased arterial tension.

CIRCULATION WITHIN THE DURAL SINUSES

The return venous blood from the ophthalmic veins passes through the cavernous sinuses (fig. 21). These two sinuses are connected by way of the annular sinuses with each other, so that communication

exists. From the cavernous sinuses the blood passes through the superior petrosal to the junction of the transverse with the sigmoid sinuses. This point is established early in the embryologic development (fig. 6). The inferior petrosal sinuses carry part of the blood of the cavernous sinuses, passing backward and downward; the superior sinus, which passes along the ridge of the petron, is located in the dura at its juncture with the tentorium cerebelli, a cross-section therefore being triangular. The inferior sinus, on the other hand, passes downward and slightly outward along the basilar portion of the occipital bone at its junction with the petron, and a cross-section of this sinus is semicircular. It empties into the jugular bulb, usually in the anterior or middle portion, never the posterior portion (fig. 21).

Fig. 18.—Slowly developing choke in a tumor of the cerebellum; veins enormously distended; capillaries distended; edema of the disk.

The sigmoid sinus receives the blood from the superior petrosal and lateral sinuses and empties into the jugular bulb. The lateral or transverse sinuses receive the blood from the large central sinuses draining both cerebral hemispheres, the midbrain and cerebellum, through the superior and inferior longitudinal sinuses and straight sinus. The general cerebral circulation depends, therefore, on a free circulation within the transverse and sigmoid sinuses, while on a free cavernous and petrosal circulation depends the orbital circulation. The former may be considered as the upper circulatory plane and the latter as the lower plane. The two join partly at the sigmoid and totally in the jugular bulb. It follows, therefore, that in a normally and symmetrically developed venous sinus system there is a free and easy circulation capable of marked adaptability. When one side is partially or temporarily blocked, the opposite side easily compensates.

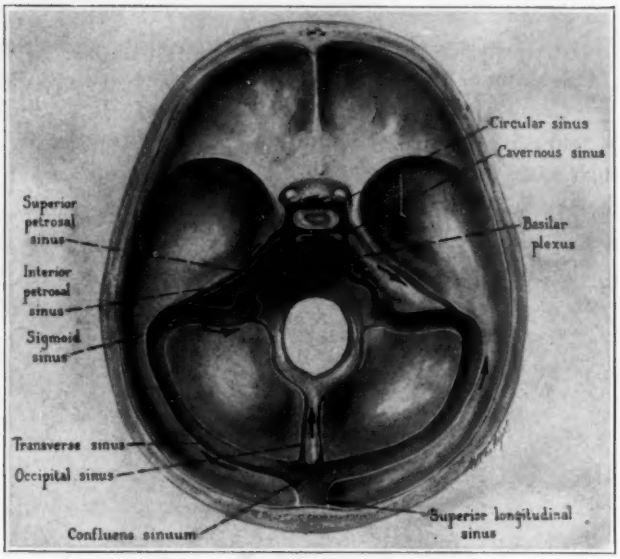

Fig. 19.—Direction of the normal circulation in the basilar sinuses.

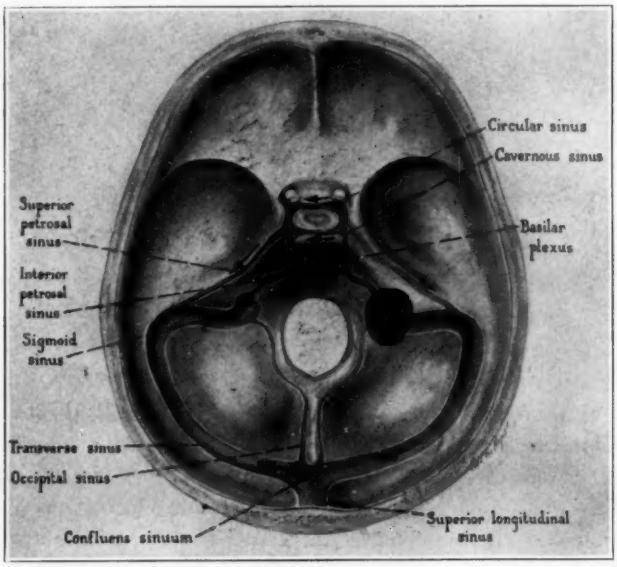

Fig. 20.—A lesion at the right jugular bulb blocks all outflow from the right side.

BLOCK OF THE SIGMOID

Under normal conditions of sinus development a block of the sigmoid sinus would cause only a partial block of the cavernous sinus of the same side (fig. 22). No appreciable stasis of the blood in the retinal veins would result because the inferior petrosal, draining into the jugular bulb, would do most of the work usually done by the superior petrosal draining into the sigmoid, and the remainder is drained through the annular sinuses to the opposite cavernous, petrosal, sigmoid and jugular bulb.

If, however, the block of the sigmoid is continued, the lateral and finally the longitudinal sinuses begin to show a stasis which eventually causes a general increase in the capacity of the opposite transverse and sigmoid sinuses. This increase may continue until such a time as either compensation occurs or a vicious cycle causes a general increase in the intracranial pressure. In general, one may say that, under normal symmetrical conditions, compression or occlusion of the right or left sigmoid sinus does not cause any change in the ocular fundus.

TRANSVERSE SINUSES

What is true of the sigmoid sinuses in symmetrically developed dural sinuses is also true of the transverse sinuses, but unfortunately the transverse sinuses are subject to marked asymmetrical development. Fully three of four cranial cavities will show a predominance of development of the right over the left transverse sinus. This is due to the coalescence of the veins of the primary plexuses in the embryo and results in the major portion of the blood from the cerebral hemispheres being carried by the right transverse and sigmoid sinuses, leaving only a small and usually central area to be drained by the straight and left transverse and sigmoid sinuses.

JUGULAR BULB

The importance of the jugular bulb in the development of the sinuses of the human being cannot be lightly valued. On this structure depends the entire success of venous return. As in case 3, the effect was incompatible with life. Lesser variations in size probably play a great part in the many conditions due to or associated with a passive cerebral congestion. Further study may reveal information of great importance along this line.

When the jugular bulb of either side is occluded, there is always at first a stasis of blood, of both the lower and the upper venous planes (fig. 20). This may be compensated by increased velocity of the blood in the opposite sinus system. A transient congestion and edema of the ocular veins results from occlusion of the petrosal inferior and

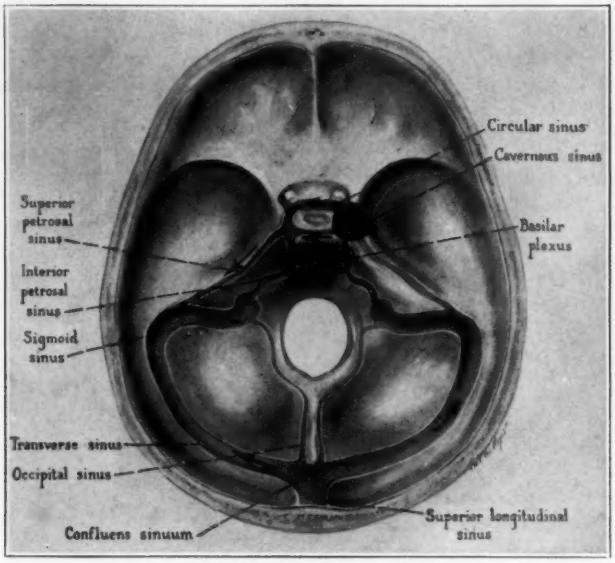

Fig. 21.—A lesion at the right cavernous sinus blocks the flow of blood from the right orbit only.

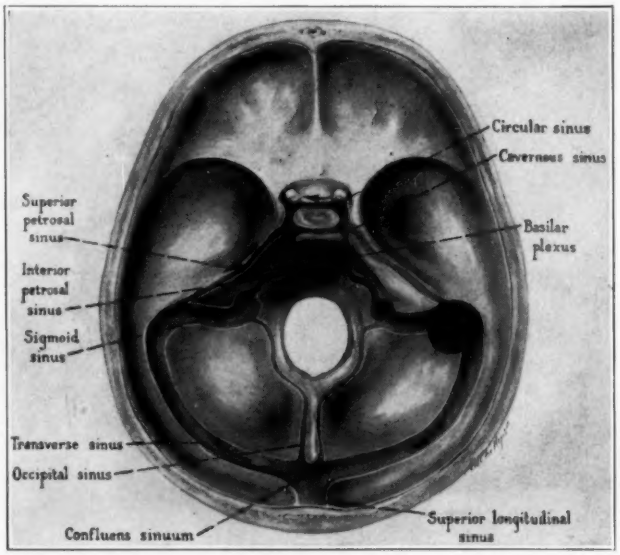

Fig. 22.—A lesion at the junction of the right transverse and sigmoid sinuses blocks the flow to the right jugular bulb except by way of the inferior petrosal sinus.

superior, while a general increased pressure is caused by the stasis in the transverse and longitudinal sinus.

RESULTS IN ASYMMETRICAL CASES

It is only when an asymmetrical development of the venous dural sinuses occurs that the compensatory flexibility of the entire venous system is apparent (figs. 1 and 2). In such cases the appearance of choked disks offers a sign of the greatest importance, while the absence of such a sign may easily be misinterpreted in the normal symmetrical development.

Since the constancy of the cavernous and petrosal sinuses is clearly established, it follows that the variations in the presence of choked disk in lesions of the cerebellar fossa must be caused by variations in the transverse and sigmoid sinuses and jugular bulb, if, indeed, the mechanical theory as to the etiology of choked disk is accepted. These variations may cause increased stasis, first, by direct occlusion of blood by way of the petrosals to the jugular veins and, secondly, by indirect pressure on the annular sinuses and petrosals.

DIRECT PRESSURE

Only a tumor mass pressing in the region of the jugular bulb (fig. 20), as in an angle tumor, can cause complete occlusion of the bulb. If the right side happens to drain practically all the blood from the superior longitudinal sinus, the occlusion will cause a rapid double choked disk, brought about by increase in the intracranial pressure. This general increase in the intracranial pressure causes a stasis of the blood in all the dural sinuses, including the annular sinuses from pressure above. In such a case, the indirect pressure added to the direct pressure from the occlusion of the petrosals causes a fulminating type of choked disk, as in case 2 (fig. 2). If, however, the tumor mass happens to be situated at the angle on the left side, in which the transverse sinus has not developed (fig. 1), or in cases in which the jugular bulb on that side is not fully developed, little or no reaction can occur until the tumor mass reaches such a size as to displace sufficient of the cerebrospinal fluid from the cisterns to cause pressure against the fourth ventricle, which, in turn, causes an internal hydrocephalus. In such a case, choked disk would be of late development, while the symptoms of cerebral pressure would be an early development. This then, would assist in the differentiation between the two cases. Case 1 is an illustration of this point.

Tumor masses situated above the tentorium, as in case 2, and pressing against the superior longitudinal sinus, can cause symptoms and choked disk of the same fulminating character as though the tumor were situated at the angle on the right side, except that in case 2 the

fulminating choked disk occurred as a result of general pressure rather than as the result of a local pressure at the bulb partially or completely closing the petrosals. Tumors situated higher in the occipital region or in the parietal region only indirectly affect the flow in the transverse sinuses and that only when the tumor mass has reached sufficient size as to cause displacement of the cerebrospinal fluid, leaving in the cranial cavity practically nothing but brain tissue and tumor, or in cases in which the aqueduct or the foramina of Monro are blocked and the cerebrospinal fluid accumulating in the ventricles acts as a tumor mass.

SUMMARY

Embryologically, the ophthalmic veins and the cavernous sinuses develop from the anterior primary plexus and the original head vein; then the primary head veins disappear. The petrosal sinuses develop from the vein draining the middle primary plexus, first almost disappearing, then enlarging in size as the future superior petrosals. The inferior petrosal, the superior longitudinal sinus and the transverse sinus develop from the anterior and middle primary venous plexuses. The lateral or transverse sinuses and sigmoid sinuses are newly developed blood vessels which form first as a communicating vein between the anastomosing anterior, middle and posterior venous plexuses and the primary jugular bulb. As the plexuses disappear and the channels coalesce, the transverse sinus fully develops and migrates, the sigmoid portion of the sinus migrating to the extreme lateral side of the developing cranium in the region of the posterior mastoid. Anomalies of the cavernous and petrosal sinuses are extremely rare. Anomalies of the superior longitudinal sinuses (bilateral sinuses) and anomalies of the transverse sinuses (size, course and location) are common. The superior longitudinal sinuses in many instances drain indirectly into the right transverse and sigmoid sinuses; the straight sinus into the left transverse and sigmoid. The transverse and superior petrosal sinuses are triangular; the inferior petrosal and sigmoid are semicircular and subject to direct and indirect pressure. Occlusion of the jugular bulb does not cause choked disk in a normally symmetrical development of the sinuses but a marked choked disk, unilateral at first and then bilateral whenever there is an absence of the left transverse sinus or a marked decrease in its caliber. Choked disk does not develop in lesions of the cerebellum and angle on the left side in those cases in which there is not a true symmetrical development on that side. Tumors situated in the cerebral fossa do not cause occlusion of the transverse or sigmoid sinuses except by indirect pressure and then only when the cerebrospinal fluid cushion has been practically obliterated and tissue presses against the sinus walls, causing first a stasis and then a collapse.

REPORT OF CASES

CASE 1.—*History.*—Mrs. D., aged 76, a widow of twenty years, was first seen by me at autopsy. Later the following history was obtained from Dr. Minnie Burdon, who was called in a few days before death. The patient had had the mild diseases of childhood but no serious illness. She had had three children, all normal deliveries. There was no record of any menstrual abnormalities. The menopause had been uneventful, occurring when the patient was about 50. About this time she began having infrequent attacks of loss of consciousness, without convulsions. These increased in frequency and severity. At about 60 she had typical grand mal, with three to four convulsions daily for several days. Attacks recurred several times monthly. At this time mental deterioration set in, with loss of memory and change in habits.

Examination.—Aug. 6, 1927, the third day of one of these attacks, the patient was examined when in bed, unconscious, with deep labored breathing and foul breath. The pulse rate was 60 and the blood pressure 180 systolic and 120 diastolic. August 7, she answered questions incoherently. She was given phenobarbital and bromides and had no more severe convulsions until July, 1928. She was seen again on Aug. 1, 1928, in status epilepticus, which terminated on August 7.

The past history and the history of the attacks were obtained with difficulty from the daughter, who was a Christian Scientist.

Autopsy Observations.—The body was rather emaciated and without scars. The calvarium was removed in the usual manner, the dura opened, and the brain removed. On removal of the brain a large cystlike mass was found on the left cerebellopontile angle. It was adherent to the dura at that point, so that on removal the tumor mass itself was left attached to the dura, while the cystic cavity remained in situ in the brain.

The brain itself showed narrow convolutions with thick sulci, as in typical idiopathic epilepsy. Large accumulations of fluid or subarachnoid lakes were noted, particularly along the rolandic fissures on both sides. The ventricles were approximately of normal appearance. The brain was kept for hardening.

Examination of the dura showed that the right transverse sinus was well developed and about twice the normal size, while the left transverse sinus did not show any development, a mere strand joining the torcular with the jugular bulb. The inferior petrosal sinus drained into the jugular bulb, but in the region of the bulb there was considerable pressure atrophy. The jugular foramen was very small, only about one-half the size of the corresponding jugular foramen.

The diagnosis was acoustic nerve tumor with cystic degeneration, left, and anomalous development of the left transverse and sigmoid sinus.

A portion of the skull was removed, showing the course of the superior longitudinal sinus into the right transverse sinus.

CASE 2.—*History.*—B. C., a girl, aged 21, complained of pain and tenderness in the back of the neck, headaches, vomiting, and peculiar sounds in the right ear, similar to the singing of a wire, of two weeks' duration. She had been operated on for acute appendicitis at Butte, Mont. About a year later she had had so-called intestinal influenza. She had then been fairly well until Aug. 14, 1928, when severe headaches commenced with pain in the head during the morning. The symptoms began with a stiff neck, pains in the back of the head, and vomiting. Menstruation was normal. There was no dizziness, and hearing, taste and smell were normal.

Examination.—There was hyperesthesia over the right side of the face to light touch, and a central paralysis of the seventh nerve on the left side; the tip of the tongue deviated slightly to the left. The patient showed inability to rotate the right eye to the extreme right position, and nystagmus, lateral in type, fine to the right, coarse and slow to the left. She could rotate the head in all directions. There was tenderness on pressure over the right mastoid tip; the grips were equal, and motor power in the legs was equal; there was no swaying or falling, and no past pointing or ataxia. The reflexes were all present and equal. Clonus was absent. The fundus showed double choked disk, equal on the two sides.

Direct ventricular puncture was done, August 21. The patient died. A preoperative diagnosis of cerebellar or pontile tumor had been made.

Autopsy Observations.—The calvarium was removed in the usual manner. The entire dura was seen to be quite bluish, as a result of a subdural hemorrhage. There was slight bulging over the right occipital lobe. The dura was opened and the brain removed. There was marked, rather extensive hemorrhage over the entire surface of the right cerebral hemisphere, more pronounced at the base than on the cortex. The convolutions were flattened on the two sides to about an equal degree. As the brain was removed, a portion of the tumor was left attached to its original site, the dura in the region of the right transverse sinus. A tumor mass about the size of an orange was found to occupy the posterior portion of the temporosphenoidal lobe and the lateral portion of the occipital lobe on the right side. The tumor was rather necrotic and into it had occurred a marked hemorrhage. The inferior surface of the brain showed the pons to be shoved to the left, causing stretching of the left seventh nerve. Section of the brain showed the left lateral ventricle to be collapsed, as a result of this distortion, in a direction from the right posterior to the left anterior areas. The right ventricle was entirely collapsed, owing to the growth of the tumor, the walls of the ventricle being practically occluded.

The dura was stripped from the base, revealing the following conditions: The superior longitudinal sinus was normal in appearance. The right transverse sinus reached from the region of the torcular much higher than the left and was approximately 1 cm. in diameter. The left transverse sinus emerged at a point 2 cm. lower and was a mere ribbon, practically free from blood. Into it, at the sigmoid junction, entered the left petrosal sinus, likewise free from blood, while the right petrosal was rather distended and entered at a point under the origin of the tumor.

The skull showed a deep groove for the right transverse sinus and no groove for the left. No occipital sinus of any size was found.

The anatomic diagnosis was: (1) right occipital endothelioma, probably originating from a vessel of the dura; (2) hemorrhage into the tumor mass and subdural on the right hemisphere.

Microscopic Appearance.—The tumor was composed generally of cells of rather large size which gave it a cellular appearance, although the cells were quite well separated generally. The intrinsic structure consisted of a radial arrangement of dark staining cells, which, in the usual section, were spindle shaped, centering about blood vessels. From the end of the cell nearest to the vessel, a cell process extended to the vessel wall, where it was apparently mingled with similar processes from other cells forming an adventitial network.

Stained by gold sublimate, these processes were dimly seen; no fibrils extended from the other pole. The cellular characteristics were those of rapid growth and high malignancy.

The tumor was diagnosed as astroblastoma.

ABSTRACT OF DISCUSSION

DR. A. W. ADSON, Rochester, Minn.: Dr. Swift has given us some interesting facts regarding the etiologic factors of choked disk, and has also called attention to a number of important points regarding obstruction of the transverse sinus. I wish we could say that choked disk is due specifically to one or the other of various factors. It is true that the author emphasized the importance of hypostatic pressure, while other investigators have shown evidence that venous stasis does produce choked disk and that malignant hypertension produces choked disk. Probably, then, all we can say is that choked disk might be due to one of two or three factors or to a combination of factors. Again, with the experiences detailed here, one becomes more or less bewildered as to the cause of choked disk and the relation of the lateral sinus to choked disk. I can recall having exposed and resected an extensive endothelioma arising from the region of the transverse sinus and associated with choked disk, and on complete resection of the sinus down to the sigmoid, the choked disk disappeared. Of course one might say that the opposite side was functioning. I think that there are certain fundamental principles we encounter, and one is that if there is a tumor that obstructs the normal flow of the cerebrospinal fluid in the region of the aqueduct, the choked disk develops rapidly; while tumors of the anterior fossa that one might even suspect of compressing the ophthalmic vein rarely cause choked disk. The olfactory group of tumors rarely cause choked disk until late. I think we ought to congratulate Dr. Swift on calling attention to this plausible explanation, and if we learn nothing else it might teach us to be on the lookout for explanations of choked disk as it develops, with the idea that possibly a certain percentage are due to tumors that might be removed.

DR. WALTER I. LILLIE, Rochester, Minn.: Dr. Swift has presented an excellent paper showing how an anomalous venous sinus plus a brain tumor can produce a choked disk. But these are selected cases and, needless to say, the anomalous sinus is not present in all cases of choked disk; therefore one must look farther for the real cause of such a phenomenon. Numerous theories have been advanced, but still clinical problems present themselves daily which cannot be explained by any one or all the present theories. In my experience, Behr's theory solves more of the clinical problems than any other. Many cases of venous sinus involvement secondary to mastoid disease have been observed without fundal changes, while one case in which both internal jugular veins were ligated did not reveal any pathologic changes of the optic disk. If choking of the disks was due to involvement of the venous sinus, it would be difficult to explain a unilateral choked disk due to an orbital tumor, or a basal frontal lobe tumor, such as described by Foster Kennedy and myself.

DR. GEORGE W. SWIFT, Seattle: It was not my intention to discuss the entire subject of choked disk but merely the part played by the transverse sinus in its development and the anomalies of this sinus. According to the Monro-Kellie doctrine, the skull can be considered as a closed cavity in which there are circulating fluids. An increase in volume of one of the three constitutent parts, namely, brain, cerebrospinal fluid or blood, must be compensated for by a decrease in the other two. In an expanding intracranial lesion there first develops, according to the Monro-Kellie doctrine, an increase in the velocity of the circulation of the venous blood. When the tumor mass has displaced sufficient blood, cerebrospinal fluid is displaced and on the location of the tumor depends the question of whether or not cerebrospinal fluid can be displaced. As Dr. Adson points out, a tumor shutting off the aqueduct of Sylvius prevents the displacement of cere-

brospinal fluid from the ventricles and this fluid acts as a tumor. Sudden closure of the cerebrospinal fluid outlet may cause a sudden increase in pressure incompatible with life. This may explain some of the sudden deaths occurring in brain tumors. Ball valve action may cause a choked disk at one time and later release of the pressure on the aqueduct may cause recedence of the choked disk. Dr. Adson's description of the ball valve action of ventricular tumors may explain some of the mechanical factors involved in choked disk. Dr. Lillie's statement that this explanation does not apply in the case of tumors of the olfactory nerve or of the acusticus is in my opinion not sound. Whenever the Monro-Kellie doctrine ceases to apply, and this occurs when the tumor reaches such size that no further displacement of cerebrospinal fluid can occur, it follows that blood entering the cranial cavity by way of the ophthalmic veins must be obstructed and a simple edema of the disks follows. Dr. Lillie states that one half of the cases of acoustic tumors do not cause choked disk. According to Cushing's early statistics, practically every case of acoustic tumor was associated with choked disk or optic atrophy. The diagnosis is made much earlier now than at the time Cushing published his monograph. All acoustic nerve tumors eventually produce a choked disk except in those cases in which the transverse sinus is asymmetrically developed and the tumor is on the side having a blind sinus.

BILATERAL COLOBOMA OF THE OPTIC NERVE

ASSOCIATED WITH HOLES IN THE DISK AND A CYST OF THE OPTIC SHEATH *

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From an ophthalmoscopic examination of what is commonly termed coloboma of the optic nerve, it is difficult to tell whether the developmental defect is in the optic nerve or in the adjacent choroid.

In a review of the literature, one encounters difficulty in classifying the cases, for there is wide latitude in many of the reports. Thus one finds the extremes—holes in the disk and extensive physiologic cup, to coloboma of the nerve head in association with coloboma of the choroid, ciliary body and iris. It is therefore impossible to tell accurately how many reports have been made, although one may have an idea from Crampton's [1] review in 1912, when he collected twenty-one cases in sixteen patients, which report included the six unequivocal examples recorded by Coates,[2] in all of which he had made histologic examinations. I found twenty-six additional cases (some very uncertain) with only one histologic report (Rochat,[3] 1913).

True coloboma of the optic nerve, therefore, is one of the rarest of congenital anomalies, and a correct diagnosis cannot be made without a histologic study, and even then there is often confusion. A clinical and microscopic description of such a case may therefore be of interest.

REPORT OF A CASE

History.—B., a female infant, aged 10 months, was brought to me for an opinion concerning a bulging of the right eye, which had been present since birth. There was no family history of abnormalities of the eyes or other body structures. The mother had three normal pregnancies, and the patient's birth was normal. A general physical examination showed that the child was normal and well developed, except for the eyes. The Wassermann reaction of the blood was negative. Examination of the eyes showed that the right eye was proptosed downward and forward. The external structures were normal, and the globe could only be rotated upward. The right orbital ring was definitely larger than the left, and an orbital growth was detected by palpation through the upper lid. The finger

* Submitted for publication, Sept. 9, 1929.

1. Crampton, G. S.: Two Cases of Binocular Coloboma of the Optic Nerve in the Same Family, Tr. Am. Ophth. Soc., vol. 13, p. 451.

2. Coates, G.: The Pathology of Coloboma of the Nerve Entrance, Roy. London Ophth. Hosp. Rep., vol. 17, p. 178.

3. Rochat, G. F.: Disease of Secondary Vesicle, as a Cause of Congenital Malformations, Klin. Monatsbl. f. Augenh., 1913, p. 444.

tension of the globe was normal. The pupil reacted promptly to a direct and consensual stimulation, and it was not definitely established that the eye had vision. The external examination of the left eye was normal, including finger tension, motility and pupillary reaction, and it was well established that vision was present. Both pupils dilated promptly and evenly on the administration of 2 per cent homatropine solution, after which a fairly satisfactory ophthalmoscopic examination was made, and the observations were later confirmed when the patient was anesthetized. In the right eye the media was clear; the disk was slightly oval in the vertical meridian and larger than normal. A large scleral ring was

Fig. 3.—Orbital cyst, producing displacement of the globe.

present; it was not so sharp as shown in figure 1, but feathered out into the adjacent choroid. A fairly dense pigment crescent lay to the temporal side. The floor of the nerve was excavated as much as 3 mm. The excavation was not so marked in the upper as in the lower part, which gave one the impression that the nerve had been hinged back. It was of dirty gray color and free from vessels. On the floor were several small dark grayish markings; a larger one on the lowest half of the disk, extending to and possibly beyond the scleral ring, was a semi-elliptical excavation of uncertain depth—literally a hole or cavern in the nerve head. Several vessels of normal size bent over the rim in true glaucomatous style, but were immediately lost from view. The retinal branching was regular, and no abnormalities were seen in the chorioretina or macular region.

Fig. 1.—Coloboma of optic disk (right) with large hole in nerve head.

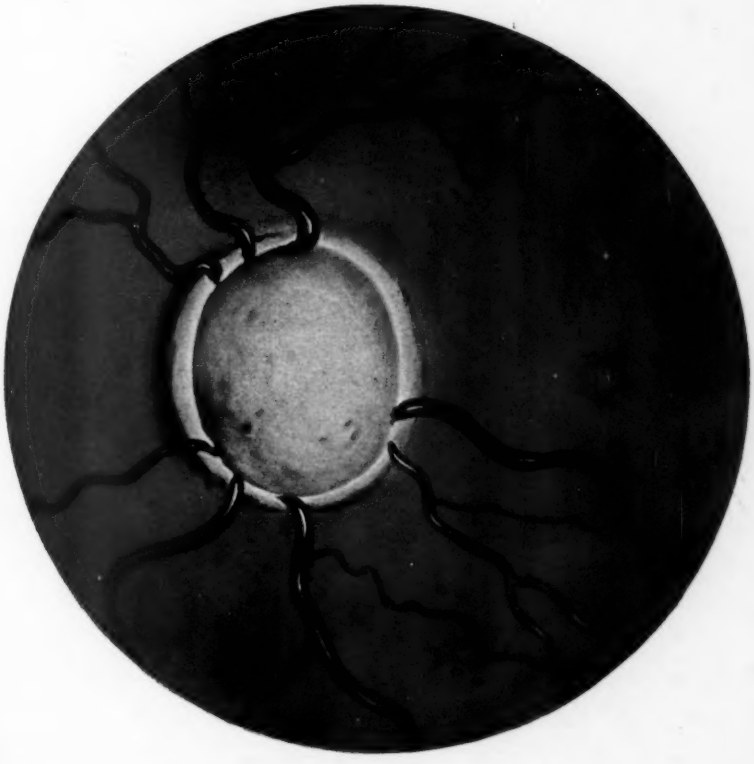

Fig. 2.—Coloboma of optic disk (left).

Fig. 4.—Globe and cyst attached, showing fragment of optic nerve.

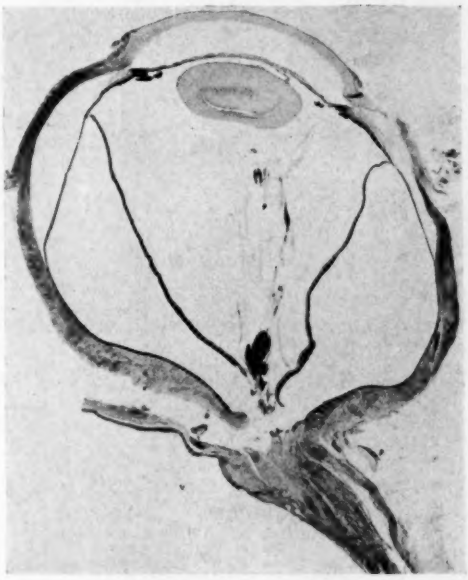

Fig. 5.—Vertical section of the globe and optic nerve. A fragment of the cyst wall (*a*) was artificially detached from the adjacent sclera (*b*). The retina and embryonic detritus has been artificially detached out of the cavern in the nerve (*c*).

Fig. 6.—The disk, cavern and attached nerve at a different level: cyst wall (*a*) detached from sclera (*b*); the cavern containing retinal elements (*c*); dural wall of nerve (*e*); intervaginal space (*f*); fragment of central vein (*g*); dilated spaces containing blood (*h*); cyst wall composed of dura (*i*).

Fig. 7.—The disk and nerve (high power).

The left fundus picture was similar, except for the absence of the markings on the disk. There were eight vessels at the disk margin.

A roentgenogram of the skull showed an enlarged right orbit, with a mass of soft tissue filling the upper part. The optic canals were normal.

The fundus picture was that of bilateral coloboma of the optic nerve, and it was concluded that the proptosis of the right eye was caused from either a cyst of the optic sheath, a meningocele or a tumor, the first being most likely on account of the congenital defects in the optic disk.

An exploratory operation was advised, with the understanding that the eye might be removed.

Operation.—Extensive external canthotomy was performed with the patient under ether. The conjunctiva over the external rectus was divided horizontally,

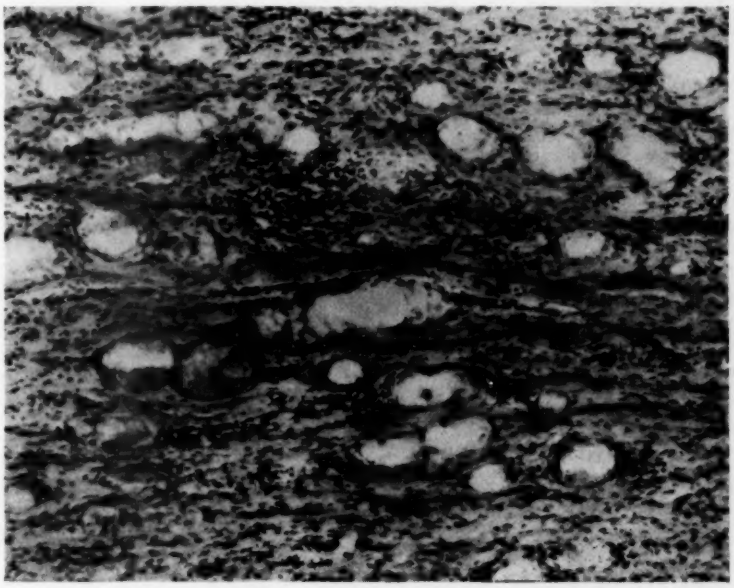

Fig. 8.—Dilated spaces in nerve, lined with endothelium and partially filled with blood (high power).

and the thin muscle was severed at its attachment and a suture applied. A large bluish-black mass which almost completely filled the orbit was exposed and found firmly attached to the globe. Sufficient fluid was aspirated for an examination in order to rule out meningocele. The immediate report was: "Benedict's solution, no reduction. Albumin, negative. Globulin, 4 plus. Microscopically, no cells or crystals." Having then determined that a cyst of the nerve sheath not containing cerebrospinal fluid was being dealt with, the remaining recti muscles were severed and the intact cyst was easily cut away from its attachment at the optic foramen. The lids and conjunctiva were repaired, and recovery was prompt.

I have followed up the case for three years. The child is growing normally, and there is every evidence that there is useful vision in the left eye. The fundus picture is unchanged.

Macroscopic and Microscopic Examination.—The contents of the cyst were withdrawn, and measured 12 cc. The fluid was straw-colored, and the report made at the time of the operation was confirmed. The cyst was injected with a diluted solution of formaldehyde U. S. P. (1:10) to distend its collapsed and very thin walls, and the entire specimen placed in the same solution. There was an unnecessary delay in sending the specimen to the laboratory and when received, the cyst and the globe had partially collapsed from overfixation.

The globe measured 21 mm. horizontally, 20 mm. vertically and 22 mm. anteroposteriorly. The diameters of the cornea were 11 mm. vertically and 12.5 mm. horizontally. The cyst measured 28 mm. horizontally and 30 mm. anteroposteriorly. Its walls were ectatic in some areas and it was firmly attached to the globe.

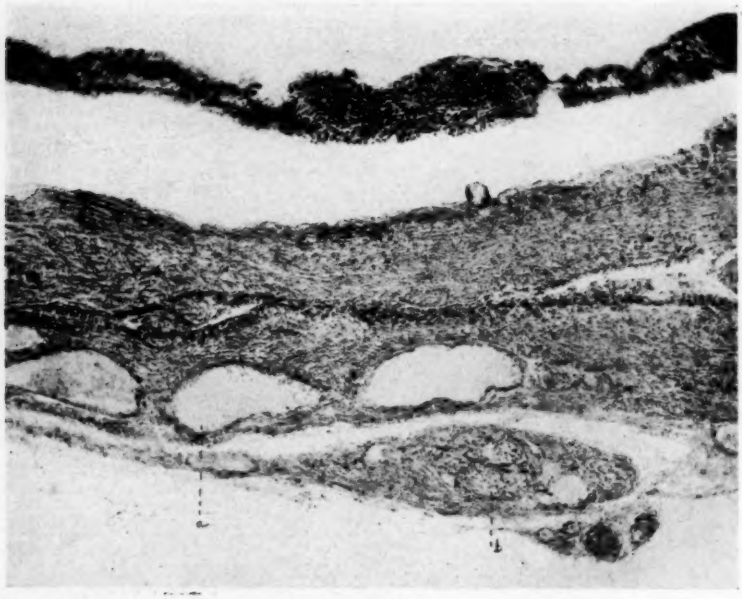

Fig. 9.—Cross-section of cyst, midway, showing dilated vessels in dural covering (*a*) and nerve fibers (*b*).

For purposes of embedding, the cyst was separated 7 mm. behind the globe, and the globe was bisected in its vertical diameter. No open channels of communication were found between the globe and the cyst, either macroscopically or microscopically. At the extreme blunt posterior end of the cyst was seen a thin stump of a severed optic nerve.

The cornea was normal throughout. The anterior chamber was deep, the filtration angle was open, Schlemm's canal was patulous and did not contain blood. The iris and ciliary body were normal. The pigment layer of the iris in the upper half was partly detached, with an attachment to the underlying anterior capsule of the lens. No inflammatory cells were found. As the pupil had dilated evenly on the administration of homatropine before the operation, this apparent posterior synechia was attributed to improper fixation.

The lens was normal with the exception previously mentioned. The sclera was normal, and the ring did not appear to be enlarged. The superior scleral promon-

tory was in normal position and the adjacent dural sheath was naturally attached. The lower promontory hung free in certain sections, and there was an attached part of the wall of the cyst (dura) for more than a disk diameter. The posterior ciliary nerves penetrated in a normal manner. Close to the nerve the small ciliary vessels could be followed into the globe.

The choroid was normal and extended nearly up to the margin of the scleral ring. There was no abnormality of that part of the choroid adjoining the inferior scleral promontory immediately above the cyst or pouchlike excavation of the disk.

The retina was completely detached from the nerve head to the ora, giving one the impression that it had been pulled out of the depressed disk. At this part the tissue was found quite altered, e. g., an absence of nerve fibers and swollen and irregular areas in the nuclear layers, some of which might have been due to improper fixation.

The vitreous was normal, except for delicate streaks of blood, which probably occurred at the time of the operation.

The scleral part of the optic nerve was present only in the upper part, when it immediately commenced to slant backward (its surface being very irregular), until a large cystlike pouch formed beneath the lower border of the scleral ring. The pouch, corresponding to the hole or crater seen ophthalmoscopically on the disk, was partially filled and lined with pigment, retinal elements and débris. No definite trace of the lamina was observed. The bulbar portion of the nerve was small and undeveloped, being composed of a conglomerate mass of glial and connective tissue with only a few nerve fibers. The dural and pial coverings, as well as its arachnoid spaces, were fairly defined in the upper part of the nerve; but in the lower part the pia was thickened, altered and welded into the connective tissue strands of the nerve. This pial covering below formed the inner wall of the very large cyst, the outer wall of which was composed of dura. A large central vein filled with blood was seen in the upper part of the nerve. Between the vein and the pial sheath above were many cystlike spaces lined with endothelium, and most of them contained blood. They were more numerous near the globe, and became fewer and smaller as the nerve grew thinner in its backward course. They were considered as varicosities or communications of the central veins.

The cyst was attached to the globe for only a small area. Its wall was composed of dura, reinforced above by a thin optic nerve. Elements of arachnoid were found in its lining. Cross-sections of the wall, made about midway, showed in one place strands of nerve fibers and several large vessels.

COMMENT

The ophthalmoscopic appearance of the case here described does not greatly differ from some of the others reported. While holes or craters in the disk have been noted in association with coloboma, it is the exception. The general location of the dark disk markings (holes) is well below the horizontal diameter, which is in keeping with other descriptions. These craters, generally described as holes, are in fact cystic defects in the mass of nerve tissue behind the level of the papilla. Here, as in the case described by Coates, the cavity contained fragments of altered retinal tissue and some pigmentation, suggesting pigment epithelium. Such defects in the nerve are generally regarded as being due to an atypical development of the neural portion of the secondary

optic vesicle, in which there is an alteration or differentiation of the layers which should properly be found within the globe. The location of these holes, that is, within the lower half of the disk, especially in association with coloboma of the nerve, strongly suggests the same developmental cause.

The arrangement of the blood vessels, seen ophthalmoscopically as they emerge from the margin of the disk, corresponds to the third grouping of Caspar's [4] widely quoted classification; that is, the vessels appear at the edges around the circumference of the disk. They are regarded as retinociliary vessels. A trace of these vessels penetrating the sclera, adjacent to the nerve, may be seen in serial sections, but no connection with its parent central vessel could be found. The central artery is not within the orbital portion of the nerve, but the vein is well placed, and the numerous cystlike spaces which are lined with endothelium and partially filled with blood are considered to be distorted branches (varicosities) of the central vein.

As has been intimated, it is difficult to differentiate clinically between coloboma of the optic nerve head and coloboma of the choroid adjacent to the nerve head. The same difficulty is often found histologically from an obscure relation to the surrounding structures. However, two important points suggested by Coates should be considered: first, the relation of the intervaginal space to the ectasia (coloboma) and second, whether or not the nerve contains the central vessels.

Should the ectasia come into relationship with the intervaginal space from the inner (neural or pia) side, it must be a coloboma of the nerve. Likewise, if the ectasia comes from the outer (dural) side, it would be coloboma of the adjacent choroid. There may be exceptions.

With regard to the central vessels in the nerve, their absence would be proof of an atypical development, yet the nerve may have otherwise developed properly from a normal closure of the fetal cleft. On the other hand, the vessels in the mesoblast may have entered in the usual way, yet the cleft failed to close.

This case shows a cavity containing retinal elements (an extension of the large hole of the disk) external to and beneath the lower scleral promontory. It appears to be an exception that a coloboma at the periphery of the nerve head had pierced the scleral promontory obliquely, and in its relation to the intervaginal space it was external to the dura.

The arrested development of the nerve may have been largely influenced (1) by the persistence of an abnormal amount of mesoblast within the cleft and its subsequent failure to close, thus crowding out or atypically placing the vessels within the nerve, and (2) by the pressure from the large intervaginal cyst especially at its distal end.

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4. Caspar: Dissertation, Bonn, 1887.

When there is a disturbance of nutrition of the edges of the ocular cleft close to the entrance into the sclera, then the fissure or cleft cannot close or unite. The union then of the nerve to the globe will not be in a circular line, but with a bow-shaped line opening downward, giving the ophthalmoscopic picture of coloboma of the optic nerve and possibly of the retina. If, however, the malformation further encroaches on and distorts the mesoblast within the furrow near the bulbar end, there would be the additional malformation or coloboma in the iris and ciliary body. For that reason, I believe that the closure of the tissue of the choroid and optic nerve are independent of each other. Such a case is described by Nieden [5] of bilateral coloboma of the nerve, with associated coloboma of the iris and choroid in one eye.

What produces these arrests or defects is unknown.

It has been shown experimentally by Stockard [6] and others that any structural arrestment or developmental defect will rarely disappear, but it will likely mar the full perfection of the eye.

NOTE.—Stockard's investigation on the abnormal or arrested embryonic development in the eyes of vertebrates and mammals may give a hint of what takes place in man. He found in the development of the vertebrate eye, the first actual organ to arise in the embryo and appear as an enormous outgrowth, that if the embryo is treated at the several critical moments determined for the origin of its different structures, its structural arrestment or alteration rarely recovers from this modification, even though a normal rate of development be subsequently established. Any interruption during development or any deficiency in general developmental energy is likely to cause imperfections of the eye.

Thus when the eggs of a marine fish are developed in sea water to which has been added salt, sugar or other substances, or by subjecting the eggs to a low temperature or reducing their oxygen supply, one or both eyes may be suppressed, or either or both eyes may show all degrees of microphthalmia or maldevelopment.

The mammalian embryo, although better protected on account of its internal development, may likewise form abnormal eyes if the female (guinea-pig) is treated with ether or alcohol, which may act directly on the placental circulation.

5. Nieden, A.: Coloboma of Sheath of Optic Nerve, Arch. Ophth., vol. 8, p. 501.

6. Stockard, C. R.: The Structure of the Vertebrate Eye as an Index to Developmental Deficiencies, Am. Naturalist, vol. 58, p. 24.

CHOROIDEREMIA *

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In 1871, Professor Mauthner [1] reported the case of a man, aged 32, with the following history, which is a free translation from the German as given verbatim by the patient.

> Ever since I can remember, I know that my vision has been poor. I can see only things that are directly in front of me, but nothing that is either to the right or to the left of me, or below or above. I could always see better in the day than I could at night. As soon as darkness appears, I see practically nothing. I also notice that I can see better in the summer than I can in winter. Since recovering from typhoid fifteen years ago, my sight is somewhat poorer. I have seven sisters, all well, and one brother 26 years of age who has exactly the same kind of visual disturbance as I have. My parents saw very well, but my father's brother had poor vision. My grandmother on my father's side became blind at the age of 70.

Mauthner's first impression on listening to the patient's history was that he was dealing with a case of retinitis pigmentosa. On examining the fundus he was, however, astonished at the conditions found. Instead of the normal red reflex, the entire fundus was a whitish green. The disks were of normal color with normal distribution of retinal vessels, although of somewhat smaller size. None of the choroidal vessels were visible, excepting in the macular region where an irregular network of small choroidal vessels appeared. There were some small masses of pigment here and there. The right eye was 3 diopters myopic with 5/200 vision. The left eye had 2 diopters myopia and 10/40 vision. The peripheral fields were irregularly contracted to about 10 degrees in each meridian.

As no similar case had ever been seen before, the question of diagnosis came up. Leber [2] saw the case and classified it as one of retinitis pigmentosa with extreme atrophy of the choroid. Mauthner could not agree with this diagnosis. He reasoned that such an extreme pigmentary retinitis was incompatible with practically normal nerve papillae. He considered the history of the patient who "saw poorly ever since he could remember," together with the results of the examination, and thought that he was dealing with congenital absence of the choroids. He named the condition choroideremia. This, of course, was purely speculative, since no anatomic studies could be made.

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* Submitted for publication, Oct. 12, 1929.

1. Mauthner: Ein Fall von Choroideremia: Ber. d. naturwissensch.-med., Vereines in Innsbruck, 1871, vol. 11.

2. Leber, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1916, p. 1185.

Several cases have since been reported, and in 1908, Nettleship [3] reviewed seven of these under the heading of diseases of the retina. These cases, however, are so extremely rare and of so much interest that I thought it worth while to again review all of the cases reported in their chronological order.

CASES REPORTED FROM THE LITERATURE

CASE 1 (Mauthner, 1871 [1]).—This is the case described.

CASES 2 AND 3 (Koenig,[4] 1874).—These two cases are reported in two brothers. The older, age 20, asked exemption from military service because he could not see in the dusk and by lamplight; his sight has been thus from his earliest recollection. He was the first born of nine children, and a younger brother was similarly affected. The other seven saw quite well. The parents and grandparents had normal vision. There was no history of consanguinity. The patient read small print by daylight, but read with difficulty in dimmed light. Color vision was good. The fields were contracted from 40 to 70 degrees, but in a moderately darkened room the fields contracted to from 4 to 11 degrees in various meridians. The disks were of natural color, and the retinal vessels were quite normal in size and distribution. The reflex in each eye was white from the absence of choroidal tissue except in the macular region where choriocapillaries were still present and were of a more or less normal color. A few choroidal vessels were seen in various parts, especially above the right disk. There were a few patches of pigment lying behind the retinal vessels. The fundi in the eyes of the younger brother were almost the same as in the older brother, but the spots of pigment were more numerous, more angular and elongated. The author stated that the whiteness of these fundi strongly resembled coloboma of the choroid, only it was not limited to the lower part.

CASE 4 (Cowgill,[5] 1892).—A youth, aged 17, presented a high degree of myopia; —13 diopters in the right eye and —16 in the left. Vision with —8 diopter lens in each eye was: right, 10/200 and left, 8/200. The entire fundus of each eye was of a scleral whiteness.

There was a binocular absence of choroidal pigment and vessels except a rich plexus forming a nearly circular patch the size of a disk diameter in the macular region and supplied by large cilioretinal vessels that emerge from the sclera, one in the right eye and two in the left, close to the temporal part of the disk. No familial history is given, and no mention is made of nyctalopia.

CASE 5 (Bullar,[6] 1898).—A youth, aged 16, when first seen in 1892, presented myopia of 5.5 diopters in each eye. After correction, vision in the right eye was 6/18, and in the left 6/12. He had always had poor vision. No note was made as to night blindness. The fields consisted of small central areas of less than 10 degrees and a peripheral part about 10 degrees wide lying between the 50 and

3. Nettleship, E.: Roy. Lond. Ophth. Hosp. Rep. **17**:373, 1908.

4. Koenig: Zwei Beobachtungen von mangelhafter Entwickelung der Choroides verbunden mit Hemeralopia, Inaug. Diss., 1874, p. 17.

5. Cowgill, Warwick W.: Report of a Case in Which There is Binocular Absence of the Choroid, Showing a Rich Plexus of Cilio-Retinal Vessels Supplying the Region of the Yellow Spot in Both Eyes, Arch. Ophth. **21**:105, 1892.

6. Bullar, O. T.: A Case of Deficiency of the Choroid. The particulars were furnished by Dr. Bullar to Nettleship and reported by him.

60 degree circle; in the right eye the field formed a complete zone; in the left eye, it formed a crescent occupying the lower and outer quadrant for about 100 degrees of the circle. The greater part of the fundus was grayish white with scattered spots of pigment. The red reflex was limited to a small patch in the macular region and around the disks. There were some isolated choroidal vessels at the periphery where there was a faint red tinge. The patient had five brothers and two sisters. Two brothers besides himself had had poor vision but were not examined.

Case 6 (Thompson,[7] 1898).—A youth, aged 18, from infancy was noticed to hold objects very close to his eyes and to peer around at anything to which his attention was directed as though he saw only straight in front of him. The other children in the family were said to have normal vision. The parents' sight was good. A granduncle was said to have had eyes similar to the patient's. The parents were not related. There was no specific history. Vision in the right eye was 20/200 and in the left 20/200. Myopia of 3 diopters was noted in each eye, and when this was corrected, the vision was 20/30 in the right eye and 20/30 in the left. The patient read Jaeger's test type no. 1. He had marked night blindness. The pupil reflexes were normal. Ophthalmoscopically, the fundi presented throughout the appearances of dazzling white sclera with the retinal vessels coursing over, as seen in coloboma of the choroid. No choriocapillaries or red reflex were seen except at the macular regions. A few small isolated vessels were scattered here and there (ciliary?). No gross pigmentation was visible. The fields of vision contracted to less than 10 degrees.

Case 7 (Otto Rosenbaum,[8] 1900).—A case of binocular retinitis pigmentosa with albinotic fundus was reported in a patient, aged 30. The vision in both eyes was 8/18.

The original of this report is not available, but Nettleship[3] included this in his review as a probable case of choroidermia.

Case 8 (Landman,[9] 1905).—A man, a brunette, aged 44, had always been well but vision had been poor during his whole life. He had experienced great difficulty in orientation and in passing from and into places of different degrees of illumination. His father never wore glasses to read, even at the age of 75. The pupils were small and reacted slowly to light. The tension and the muscle movements were normal. The irides were normal and deep blue. The color sense was normal. The fields showed an irregular concentric contraction to about 3 degrees. Vision in the right eye was 6/18. The patient read Jaeger's test type no. 3 at ten inches. With —1 diopter cylinder axis 90, the vision was improved to 6/8. Vision in the left eye was 6/18. With this eye the patient read Jaeger's test type no. 5 at ten inches. There was no improvement in distance vision with lenses. He read Jaeger test type no. 3 when +2 diopter lens was added. Ophthalmoscopic examination of the right eye showed a brilliant white reflex. The sclera showed through everywhere; the perfectly white background was relieved only by the presence of the pale disk with its normal central vessels and a vascular plexus in the macular region. Here and there scattered about the fundus were small thin masses of pigment about the size of a half pinhead. No choroidal vessels were anywhere visible. The central artery and vein were very minute and underdevel-

7. Thompson, J. Tatham: Congenital Deficiency of the Choroid, Tr. Ophth. Soc. U. Kingdom **19**:140, 1898.

8. Rosenbaum, Otto: Ueber Retinitis Pigmentosa, Kiel, 1900.

9. Landman, Otto: A Case of Symmetrical Congenital Absence of the Choroid and Retina Except in the Macular Region, Arch. Ophth. **34**:473, 1905.

oped, and soon after leaving the disk became so attenuated that in parts of the retina somewhat remote from the disks they were scarcely discernible. In the macular region was a group of small and large vessels forming a fluffy plexus and filling an area a trifle larger than the disk. From the upper outer portion of this plexus a large broad vessel in a single trunk passed obliquely upward and outward toward the periphery of the fundus, divided into two streams which united again in the form of a single vessel just before it lost itself in a plexus of vessels in the upper outer periphery of the fundus, presumably the vorticose vein. From the inner upper corner of this same macular plexus a broad vessel started upward and inward, and on a line with the vorticose vein just mentioned, sank abruptly through the sclera and was lost to view. Nasally, from this vessel was another group of vorticose veins with no vessels of any kind issuing from it or going to it. In the outer lower periphery of the fundus, another group devoid of outgoing and incoming vessels could be seen forming a vorticose vein. These two groups formed isolated islands on a white sclera. No cilioretinal vessels could be seen. The disk was surrounded by a very broad scleral ring. The description applied to the fundus of the left eye, except that here there were only two vorticose groups in the upper part of the fundus and none in the lower portion. There was also a difference in the arrangement of the blood vessels coming from the macular plexus. There were three vessels issuing from this plexus; they pursued an almost parallel course upward and outward to a venous plexus. They started as small branches which soon converged and made a thick trunk. From the outer and upper vorticose group numerous vessels passed toward the periphery and were lost to view. There were seven small faint blotches of pigment in the macular plexus.

CASE 9 (Connor,[10] 1919).—A soldier, aged 25, complained of night blindness so that he was unable to perform the duties of night guard. This condition existed as long as he could remember. His eyes had never been sore and did not trouble him when he went to school. He had formed the habit at home of not going out much at night unless some relative or friend went with him. Before entering the army, he did clerical work without trouble to the eyes. The personal history was unimportant. The patient thought that he had measles when 6 or 7 years old, but that the eyes had not been affected. He said he had not had venereal infection. The parents were living and well, and were not related. One sister and one brother were living and well. There was no other case of night blindness in the family. The patient's vision was 20/20 in each eye. The lids and conjunctiva were normal. The pupils reacted to light and accommodation. The irides were normal and dark brown. Ophthalmoscopically, the lens and vitreous were clear. The fundus was a glistening white over which the larger retinal vessels appeared about normal in size and distribution. The sclerotic was exposed to view over the entire fundus, with the exception of a circular area about twice the size of the disk in the macular region. In this macular area the appearance of the fundus was normal, having the normal yellowish red color. The macula showed as a slightly darker spot in about the center of this patch of normal fundus. Scattered about the fundus were a few small ciliary vessels and a few spots of pigment. Both eyes were affected in the same way and presented practically the same ophthalmoscopic picture, except that the left macular area was less regular in outline than the right, the latter having the choroidal vessels extending out from it toward the disk and below. The retinal vessels appeared

10. Connor, A. B.: Congenital Choroideremia, Am. J. Ophth. **2**:553, 1919. Jackson, Edward: Editorial, Choroideremia, ibid. **2**:618, 1919.

entirely normal. The visual fields were contracted to from 60 to 30 degrees. The color fields were correspondingly contracted, the red and green having a normal relation.

CASES REPORTED BY THE AUTHOR

In addition to these cases, I shall report the following two cases of patients who presented themselves for examination at the Research Hospital, College of Medicine of the University of Illinois:

CASES 10 and 11.—Two brothers, J. J. and G. J., aged 40 and 42, respectively, had had poor vision since childhood. Vision was very poor at night. The parents were not related. The father's vision was good, and the mother always had poor vision but nothing was known of the cause. Three other brothers had good vision. The personal histories were negative. The older brother had received a right parietal skull fracture one year before examination from which he remained unconscious for three weeks. He thought that his sight was worse since the injury. The two brothers had consulted several oculists, and their condition was diagnosed as "chorio-retinitis pigmentosa." Wassermann tests of the blood and spinal fluid gave negative results. The vision of J. J. in the right eye was 12/200; with —1.75 sphere it was improved to 20/100. Vision in the left eye was 20/200; with —1.75 sphere it was improved to 20/100 plus 1. The vision of G. J. was 20/65 in the right eye; with —1 sphere and —1 cylinder x 90 it was improved to 20/20. Vision in the left eye was 18/200; with —1.5 cylinder x 90 it was improved to 20/30. Each brother had a near point of 30 cm. The older brother had a slight divergence of the right eye for distance, otherwise the eye movements were normal in all directions. The pupils were 4 by 4 mm., and were round and reacted sluggishly to light. The reaction for accommodation was good. The irides were greenish blue and of normal structure. The anterior chambers, lenses and vitreous bodies were all normal. The tension was normal. The ophthalmoscopic pictures were nearly identical in all four eyes. The disks were of a normal reddish color against a greenish-white background. Except for the normal course of the retinal vessels and the macular areas, the fundi appeared almost structureless. The macular areas were red instead of the almost uniform greenish paleness. Here there was a preservation of modified retinal and choroidal structures and a few specks of pigment. This modified area extended beyond the macular areas almost to the temporal periphery. The retinal vessels were normal, the arteries somewhat contracted. Here and there in the periphery were seen a few specks of pigment, some of which were triangular and were placed on top of the retinal vessels. The peripheral form fields were concentrically contracted to less than 10 degrees. There was color perception at the point of fixation only. Figure 1 shows the right fundus of the younger brother and figure 2 the left fundus of the older brother. Figures 3 and 4 represent the visual fields in each case.

Excluding the case of Rosenbaum, which is questionable, there are altogether ten reported cases in which the ophthalmoscopic picture shows essentially an absence of the choroid except in the macular region. All of these cases are in males. When more than one in a family was affected, or as in Thompson's case, in which a remoter relative may have been affected, the affected person was always on the male side. No authentic case in a female has ever been reported. The mother of my two patients had poor vision, but nothing definite can be said as to the cause. That the cases are congenital or at least developmental in

origin, there can be no question. Nearly all of these cases presented myopia of varying degrees. The most prominent symptom was nyctalopia, for which reason Nettleship described his cases under retinitis pigmentosa although he stated that this was for purely clinical purposes.

Fig. 1.—Right fundus of younger brother.

Fig. 2.—Left fundus of older brother.

He recognized that it was improbable that there was any relation to this disease.

Since no anatomic studies have ever been made, any statement as to the probable origin of choroideremia is purely speculative. Nettle-

ship explained the appearance of patches of pigment by the fact "that the retinal epithelium is laid down in the first few weeks of embryonic life, before the choroid which is to nourish it has been differentiated; therefore, if the choroid atrophies or does not develop, we should expect that the epithelial pigment, so far as it has been formed, would retrogress and migrate."

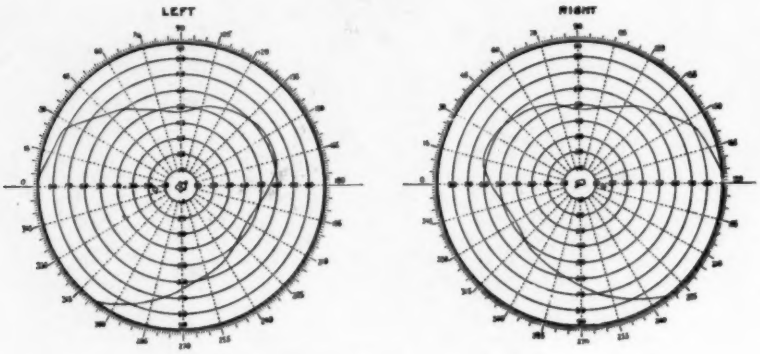

Fig. 3.—Visual fields of younger brother.

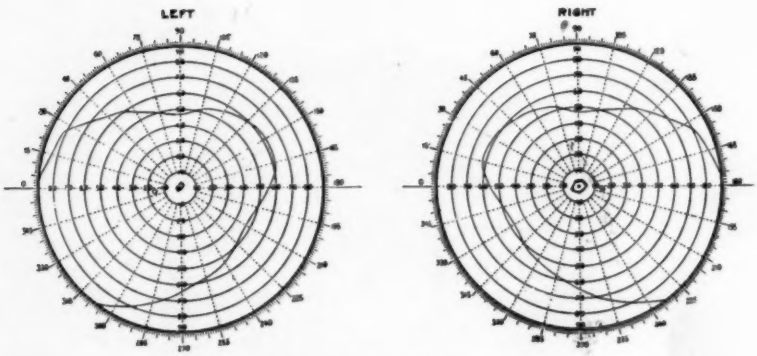

Fig. 4.—Visual fields of older brother.

Connor reviewed in detail the embryology of each structure, and stated:

The analog in embryonic life of what is later the macular area of the fundus, is a spot in the outer surface of the secondary optic vesicle where the first signs of differentiation of the mesoblast into sclera and choroid are seen, the first appearance of the choroid being manifest by a tiny plexus of capillaries at this point. Hence, in seeking an explanation of the cause of choroideremia, we must consider that differentiation of the mesoblast of the secondary optic vesicles into sclerotic and choroid began in a normal way and at the normal point, but for some reason was arrested when only the macular area was supplied with choroid.

Thus, both Nettleship and Connor ascribed the condition to an arrest in development.

Jackson [10] called attention to the physiologic importance of the choroid, and thought it almost inconceivable that a large part should be lacking in an eye possessed of standard vision in daylight. The cases so far reported, however, show definitely that such a condition may be possible. They also show the independence of the anterior uveal tract to the choroid. At least, in my two cases, neither an anatomic nor a physiologic anomaly could be demonstrated in the anterior segment of the eye. Anatomic studies in a case of choroideremia should prove most interesting both from the standpoint of anatomy and from the standpoint of physiology of the eye.

Ophthalmologic Review

THE PATHOGENESIS OF GLAUCOMA*

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While it cannot be said that the long debate over the pathogenesis of glaucoma has been brought within sight of a satisfactory conclusion, recent investigation has at least revealed some important facts, without an understanding of which any solution would always have been impossible. This is especially true of the observations in three fields: the biochemistry of the aqueous and vitreous, the physiology of the ocular blood-vascular system, including the capillaries, and the observations of the normal and glaucomatous eye which newer means of observation, such as the gonioscope and the slit-lamp, have made possible. As Duke-Elder stated,[1] it is undoubtedly true that different schools of investigation have tried to make their particular observations the whole basis of a theory of glaucoma, to the exclusion of other investigations, when each school may have elucidated factors of importance in the disease. Still, in a review of the last ten years' progress, it will be necessary to take up some of these individual theories with the evidence for and against them. The present knowledge of the aqueous and of its circulation in the eye has recently been reviewed in the ARCHIVES by Yudkin,[2] and only such points as are necessary for clearness will be touched on here.

Seidel and his school, using all three of the foregoing methods of investigation, have concluded in favor of the most widely accepted theory of glaucoma, that most cases are due to a blocking of the drainage channels for the aqueous, chiefly at the chamber angle. As a basis for this conclusion, Seidel has offered evidence that the aqueous is a true secretion of the ciliary epithelium, and that its principal avenue of drainage from the eye is by Fontana's spaces and the canal of Schlemm. This evidence was afforded by the anatomic structure of the ciliary epithelium, in which granules and vacuoles characteristic of secretory epithelium could be demonstrated;[3] the effect of drugs such as pilocarpine which stimulate glandular activity on the ciliary epithelium;[4] the appearances after vital staining,[5] and the effect of injecting toxic substances such as potassium cyanide into the vitreous, which lowers the

* Submitted for publication, Sept. 13, 1929.

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intra-ocular tension apparently by reducing ciliary secretion.[6] Seidel also investigated the circulation in the anterior ciliary arteries by means of a transparent capsule attached to a manometer, by which pressure necessary to occlude the artery could be measured. By this means he calculated that the pressure in the intra-ocular capillaries is 30 mm. of mercury, which, allowing for the intra-ocular tension of 25 mm. and the osmotic pressure of the blood, he believed was insufficient to allow fluid to enter the eye without the intervention of a true secretory process.[7]

Other evidence was afforded by the fluorescein test applied to fistulizing scars after trephining. By dropping 2 per cent fluorescein on the conjunctiva, Seidel [8] observed a clearing of the fluorescein over the bleb which was markedly increased following the application of physostigmine and decreased after the administration of atropine. He collected the fluid on gauze rolls and by weighing demonstrated these differences.

Another interesting clinical demonstration of the relation between the size of the pupil and intra-ocular tension is the recording of tension before and after a stay in the dark room. In patients with a shallow anterior chamber, Seidel [9] and later Serr [10] found a marked increase in intra-ocular tension, which rose in one case from 20 to 80 mm. within an hour, returning to normal a half hour after the patient returned to the light. This they could explain only as the result of stopping of the chamber angle when the pupil is dilated. Seidel [11] also found that the weight of the tonometer produced five times as great a fall in tension with a small pupil as with a large one, presumably because the chamber angle is open in the former case.

Feigenbaum [12] confirmed the rise of tension after dark adaptation in glaucoma, and considered a rise of 8 mm. of mercury pathognomonic of early glaucoma.

An interesting method of demonstrating drainage from the anterior chamber to the canal of Schlemm and the anterior ciliary veins was devised by Seidel [13] who made injections into the anterior chamber with a colored solution and by raising the pressure slightly above the intra-ocular tension, could observe these vessels fill with the fluid. He expressed [14] the belief that this is the only drainage channel of importance, and was not able to agree with the conclusions of Magnus [15] and Stübel [16] who claimed to demonstrate lymph channels in the iris by injecting hydrogen dioxide into the anterior chamber.[17] Seidel believes that glaucoma occurs either as a result of hypersecretion of aqueous or of insufficient drainage at the chamber angle. Work leading to entirely different conclusions has been reported by Hamburger in Germany, Magitot and Bailliart in France, Duke-Elder in England, and Yudkin in this country.

Hamburger was first led to doubt the accepted theory of intraocular circulation by a suggestion of Ehrlich, and in 1900 proposed his vascular theory.[18] His experimental work [19] on the subject was collected in 1914, and many communications on the subject followed. His position, as expressed in 1926,[20] remains approximately unchanged. It is that the basis of glaucoma is a relaxation of the vasoconstricting mechanism, causing a relaxation of the blood vessels and a repletion of the eye with blood. This action is most pronounced in the vascular choroid. The cases of acute glaucoma associated with nevus vasculosus [21] and following emotional stress illustrate this theory especially well. The experimental work brought forward by Hamburger in recent years is chiefly that afforded by the use of epinephrine and an epinephrine substitute in glaucoma, although his explanation of their effect, by converting a venous to an arterial hyperemia, has not been unanimously accepted.[22] Hamburger's original opinion, that the pupil is ordinarily impermeable to aqueous and that the iris is the principal source of the aqueous, aroused a storm of discussion, being opposed especially by Seidel. Hamburger has defended his original position in numerous articles.[21] Although his opinion is far from being generally accepted, his theory as to the cause of glaucoma does not rest or fall on these points and, as will be seen, his principal conclusions have found much support in the work of later investigators.

Magitot expressed the belief that the aqueous is stored up in the eye by dialysis from the blood, and that it has practically no circulation. This opinion is based on chemical studies of the aqueous [23] and on studies of the ocular circulation.[24] In a recent review, Magitot and Bailliart [25] summed up their reasons for believing that the primary cause of glaucoma is an obstruction in the choroidal veins. These are:

(*a*) The obstruction of the cortex veins and anterior ciliary veins experimentally produces hypertony.

(*b*) In an animal killed by bleeding, perfusion restores the ocular tension to its former level.

(*c*) Compression of the carotid and retrobulbar injections of epinephrine which remove blood from the choroidal cushion both produce hypotony.

(*d*) Injections of irritating substances (Wessely) or contusion (Leplat) which cause choroidal hyperemia and inhalation of amyl nitrite, which causes capillary dilatation, produce increase of tension.

(*e*) Section of the sympathetic nerve with resulting vasodilatation produces hypertony, and its stimulation has the opposite effect.[26]

(*f*) Reports of sections in early cases of glaucoma show inflammatory periphlebitis or sclerosis of the choroidal vessels almost uniformly. This is even true of the eyes of buphthalmic infants, and the

absence of Schlemm's canal may be the result of intra-uterine inflammation of the venous system of the eye.

(*g*) Pathologic conditions causing obstruction in the venous return from the eye, such as orbital cellulitis (Magitot [27]) tenonitis and pulsating exophthalmos, have been reported as causing secondary glaucoma.

While there is some relation between the general blood pressure and glaucoma, as is shown by the fall of tension after compressing the carotid, clinical experience teaches that it is not the general blood pressure which is usually responsible for pathologic hypertony but rather some factor causing obstruction to the local venous circulation in the eye, and this, Magitot and Bailliart believe, is primarily a vascular inflammation. That a dilatation of the local capillaries will allow the general blood pressure to affect ocular tension directly is shown by Magitot's experiment [24] in which a subconjunctival injection of saline solution is first given, followed by an intravenous injection of solution of pituitary in which case the ocular tension rises with the blood pressure, reaching 125 mm. of mercury in the cat. It is here that another factor, to which these authors also give due respect, comes into play: the nervous mechanism of the vessels. With an emotional crisis or whatever else deranges the vasomotor equilibrium, an attack of glaucoma may result but only, they believe, in eyes already possessing a more or less marked diminution in the caliber of the choroidal veins.

By a method of direct estimation of the pressure in the retinal arteries, Duke-Elder [28] found it to be 64 mm. of mercury diastolic and 88.5 mm. systolic and believed that the pressure in the ciliary vessels must be about the same. Baurmann,[29] using Seidel's method, obtained higher values than Seidel, 53.7 mm. as the diastolic pressure in the anterior ciliary vessels, a pressure high enough to allow fluid to enter the eye from the circulation without the aid of a secretory pressure as postulated by Seidel. Since Duke-Elder found the venous pressure in the eye to be about 39 mm., i. e., higher than the intra-ocular pressure, he believed that a constant circulation of aqueous out of the episcleral veins is impossible, but that slight intermittent increases in the intra-ocular tension force small amounts of aqueous out of the canal of Schlemm which acts as a safety valve.[30] A study of the chemistry of the aqueous [31] led him to the conclusion that it is a dialysate from the blood through the capillary walls. He believed that the anatomic proofs of the glandular activity of the ciliary epithelium offered by Seidel and others are not conclusive, since some animals possess no ciliary body and others a ciliary body with no evidence of glandular structure.[32] Koller [33] and Thiel [34] have shown that physostigmine causes a vasodilatation which would explain the increased production of aqueous observed by Seidel without the assumption of a secretory stimulation. Adler and

Landis [35] have also shown that this increased formation of aqueous after the administration of physostigmine persists after removal of the ciliary ganglion and hence must be due to vasodilatation. Yudkin,[36] Baurmann [37] and Tron [38] found results agreeing in the main with those of Duke-Elder and expressed the belief that the aqueous is a dialysate of the blood. Wessely,[39] examining the aqueous after paracentesis, found a difference of the protein content in men such as had been described in animals, and hence denied the claim of Seidel that there is an essential difference in the mode of aqueous production in man as compared with that in experimental animals. He believed that the aqueous is a dialysate of the blood in the whole uveal tract. The chief difference between Duke-Elder's point of view and that of Hamburger and Magitot is that he admitted [39] that the canal of Schlemm has more function as a drainage channel for aqueous though only, as previously mentioned, owing to intermittent rises in intra-ocular tension. Priestley Smith,[40] while not insisting on a secretory function for the ciliary epithelium, stated the belief that differences in pressure produce a constant circulation from the ciliary capillaries to the canal of Schlemm, upholding Leber's filtration theory. In view of his conviction that the aqueous is a dialysate of the blood, Duke-Elder did not believe that obstruction of the chamber angle is the only important cause of glaucoma. While recognizing that it may occur, he believed that such obstruction is secondary to other changes in the vitreous, the vasomotor system, etc., each of which must rather be regarded as the primary cause of rise of tension in individual cases.[41] He even suggested that physicochemical changes in the retina and nerve may be responsible for the cases of simple glaucoma without rise of tension which have been recorded.

Because of the frequent occurrence of glaucoma in elderly persons, attempts have been made to relate the increased intra-ocular tension to increased blood pressure and arteriosclerosis. Thus, Charlin [42] found that 90 per cent of patients with glaucoma were the subjects of arteriosclerotic changes.

Both Magitot and Duke-Elder, while recognizing this frequent association, stated the belief that high blood pressure itself cannot affect the intra-ocular tension unless accompanied by sudden change of pressure (Duke-Elder) or disturbances of vasomotor control which allow acute capillary dilatation (Magitot).

A number of observers have considered certain chemical changes in the blood important as causes of glaucoma. Since the ocular tension can be brought down by making the blood hypertonic, the osmotic pressure of the blood in patients with glaucoma was investigated, and Hertel and Citron [43] found hypotonicity in a majority, below 5,000 in 45 per cent of the cases, while patients with normal tension never showed pressures so low. Ascher [44] found the sodium chloride of the blood reduced in

glaucomatous patients; this would cause hypotonicity. According to Duke-Elder, however, these changes in osmotic pressure, if they affected only the salt content of the blood, could have no permanent effect on the ocular tension since the eye is permeable to salt and the aqueous would soon reach the same osmotic pressure as the blood so that the tension would return to normal. It would only be a change in the colloid content of the blood which could have a permanent effect by its osmotic pressure, and Serr [45] and Dieter [46] have found no abnormality in this respect in the blood in cases of glaucoma.

Passow [47] investigated the blood chemistry in a series of cases and found certain changes which he considered characteristic of glaucoma. These were an increase of iodine and epinephrine, a decrease of calcium in proportion to potassium and a decrease in choline. On account of the recently discovered effect of choline in lowering the blood pressure, he considered the decrease of choline especially important in the production of ocular hypertonia and was able, in part, to decrease the tension in rabbits by giving choline intravenously. He also noted in patients with glaucoma an increase in basal metabolism and an alkalosis of the blood, which he believed produced an increased tone of the sympathetic nervous system which would account for some of the phenomena observed in glaucoma.

Since the recent work of Krogh on the capillaries and the clinical observations of Muller in capillary microscopy, a number of observers have emphasized abnormalities of vasomotor control, especially in the regulation of the capillaries, in patients with glaucoma. Scheerer and his associates [48] have found by capillary microscopy that most subjects with glaucoma show enlargement of the venous part with constriction of the arterial part of the capillary loops, hemorrhages and other signs of capillary stasis which Muller considered characteristic of the vasoneurotic diathesis. A large number of patients were submitted to a subjective test described by Scheerer [49] in which the subject observes his own retinal circulation before the uviol light; most of the patients with glaucoma observed the lack of regular pulsation characteristic of the vasoneurotic diathesis.

Horniker [50] confirmed the presence of Scheerer's sign in thirty-two of thirty-six cases of glaucoma and also found in these cases numerous other signs of vasoneurosis, such as a history of asthma, urticaria and scintillating scotoma, all pointing to defective vasomotor control. He noted a difference in blood pressure as estimated on the right and left brachial artery in most of these cases, pressure being highest on the glaucomatous side in several unilateral cases. These authors deduce that the increased tension in these cases is due to an increased permeability of the capillaries, as was formerly suggested by Magitot. Further evi-

dence of this increased permeability was submitted by Schmidt,[51] who found that after drinking 1 liter of water, sixteen patients with glaucoma presented an atypical curve of blood dilution, as shown by frequent estimations of hemoglobin and urinary specific gravity. They also showed an increase in intra-ocular tension which was never seen in normal patients. This could be prevented in one eye by the previous use of pilocarpine. Römer [52] considered the results of this experiment proof of a capillary-endothelial derangement in glaucoma. Dieter,[53] using a subjective method of estimating capillary pressure in the eye, always found it increased in glaucoma.

The undoubted relation between the intra-ocular tension and the general circulation was shown by Schoenberg,[54] who investigated the rise of tension following compression of the jugular veins. He believed that the maintenance of normal tension depends on the integrity of a vascular unit within the eye, consisting of arterioles, capillaries and venules, all under reflex control. Gradle [55] suggested that three factors are probably present to account for an attack of glaucoma: (1) an anatomic structure which will allow the chamber angle to be easily closed; (2) a lack of tone of the sympathetic fibers that control the ocular vessels; (3) a sudden rise of systemic blood pressure. Goldenburg,[56] referring to Krogh's work, suggested that dilatation of the capillaries in the ciliary body causes it to swell and block the chamber angle.

While the action of some of the newer therapeutic agents such as an ergot preparation, barium and solution of pituitary, and an older one, calcium, has been explained by their lessening the permeability of the capillaries, their mode of action is still a matter of debate and the fact that another drug, choline, produces marked vasodilatation and still reduces the ocular tension indicates that not much definite evidence as to the pathogenesis of glaucoma has been afforded by their use,[22] although something may be expected along these lines with further investigation. Because of the reported effects of some drugs and glandular extracts and of the frequency of glaucoma at the time of the menopause, attempts have not been lacking to trace glaucoma to an endocrine imbalance. It may be said, however, without taking up such claims in detail, that the present knowledge of endocrine imbalance and its effects on the sympathetic nervous system and the capillary control is too incomplete to allow of any definite conclusions as to a relation between glaucoma and the endocrines.

That there is a definite nervous mechanism which is of importance in the control of tension was shown definitely by Shiraishi,[57] who noted a constant increase of tension in both eyes, but especially in the opposite eye, following destruction of the ciliary ganglion in rabbits. As pre-

viously mentioned, Magitot found the tension increased after section of the sympathetic and decreased after its stimulation, which he interpreted as due to an effect on the vessels. The work of Leplat [58] and Weekers,[59] who found that contusion of the globe (Leplat) or cauterization of the sclera (Weekers) produced an increase of tension affecting also the opposite eye, was interpreted as due to the stimulation of some vasomotor reflex which Weekers called the consensual ophthalmotonic reaction. Post [60] was able to reduce tension temporarily by cocainizing the sphenopalatine ganglion and even more markedly by making injections of alcohol into the ganglion. He referred to the cases of Wilder and others in which sympathectomy produced a fall of intra-ocular tension and credited Lagrange with first propounding the theory that glaucoma is caused by hyperactivity of the sympathetic with resulting hypersecretion of aqueous.

These indications that vasodilatation plays a part in causing glaucoma combined with slit-lamp observations in glaucoma have convinced some observers that possibly all cases of glaucoma, even simple glaucoma, depend on a primary inflammation.

After examining seventy-one patients with glaucoma with the slit-lamp, Malling [61] found that sixty-two showed signs of iridocyclitis and hence must be classed as having secondary glaucoma. By comparison with a previous series of patients examined without the slit-lamp, he found that in thirty-three of these sixty-two patients the condition would have been classed as primary glaucoma when the patients were examined by the ordinary methods. Only nine patients were seen who showed no signs of iridocyclitis, and all stages were observed between the condition in these patients and that of those with frank secondary glaucoma. Patients were seen showing iridocyclitis with increased tension in one eye and simple glaucoma in the other. In a few cases of primary glaucoma with no signs of iridocyclitis, the appearance of such signs, after a year or longer, was noted. Hence, Malling believed that a quiet iridocyclitis may be the primary cause in all cases of glaucoma.

The appearance of fluorescein in the aqueous after oral or intravenous administration, which occurs in glaucomatous eyes, as pointed out by Thiel [62] and others, has been considered evidence of an inflammation, as it occurs also in eyes with iridocyclitis, while in normal eyes fluorescein seldom appears in large enough amounts to be visible. Seidel,[63] however, who confirmed its occurrence in twenty-one cases, considered this an evidence of hypersecretion in glaucoma. Hamburger, as previously stated, believed that some inflammation was the primary cause of the venous stasis which he assumed to be the cause of glaucoma.

Not much need be said of the pigment theory of glaucoma which claimed that pigment granules from a degenerating iris and ciliary body

block the chamber angle. This theory, first propounded by Levinsohn in 1908 and brought into prominence by the slit-lamp examinations of Koeppe,[64] Schieck,[65] Gradle[66] and others, lost interest after Vogt[67] showed that pigment granules in the chamber angle and on Decemet's membrane were commonly found in eyes without glaucoma and must be considered a physiologic occurrence in old age.

Recent work on the vitreous has brought up a question which may prove to be of importance in the pathogenesis of glaucoma. In 1910, Fisher called attention to the fact that the vitreous, behaving as a colloid, swelled when brought to an acid reaction, and suggested that glaucoma might be the result of such swelling in states of acidosis. This theory was not accepted, however, in view of the clinical fact that in the extreme acidosis of diabetic coma, the eyes become soft. Baurmann[68] explained this by showing that the iso-electric point or reaction at which a colloid occupies least space is in the case of the vitreous at p_H 4.2, a reaction much more acid than ever occurs in the body fluids. Thus, even the extreme degrees of acidosis seen during life would only bring its reaction nearer this iso-electric point and hence cause actual shrinking of the vitreous. The reactions at which Fisher observed swelling were much more acid than could be reached during life. Baurmann found the reaction of the normal vitreous to be that of the blood plasma, about p_H 7.6, and thought that this corresponded to its maximal degree of swelling, so that further swelling of the vitreous could be ruled out as a cause of glaucoma. Redslob and Reiss,[69] however, found that increasing the alkalinity caused further swelling up to p_H 8.2, a definite amount of swelling following each change of 0.1 above p_H 7.7, which he considered normal. Since he found that 0.02 cc. of fluid added to the human eye at normal tension raised the tension to 60 mm. by Schiötz's tontometer, he believed that it is quite possible that changes in tissue alkalinity occurring during life might cause enough swelling of the vitreous to result in glaucoma. It is also possible, he suggested, that the iso-electric point of the vitreous may change under pathologic conditions, so that the normal tissue reactions would cause swelling and hence glaucoma.

A glance at the conflicting opinions included in this review shows that the classic view of glaucoma and the physiology of the intra-ocular fluids at present taught in most schools is somewhat too simple to account for all of the clinical and experimental facts observed. The definite evidence of change of tension with the size of the pupil and other evidence seem to leave no doubt that the chamber angle and canal of Schlemm play some part in the drainage of the aqueous, if only perhaps as a safety-valve, and that its obstruction can cause glaucoma. Evidence seems to be accumulating, however, that the aqueous is not a secretion, but a dialysate of the blood, and hence that vascular disturbances are of

great importance in its formation and in the production of glaucoma. The nervous influence on ocular tension which is undoubtedly present would seem to be mediated by its effect on the vessels, especially the capillaries. What part changes in blood chemistry may play is still an open question, but the evidence that such changes through their effect on capillary permeability are important is accumulating. Swelling of the vitreous may be found to be an important factor. Low grade inflammation is probably a factor in many cases, even of apparently simple glaucoma, producing venous stasis or increasing capillary permeability or both.

It is evident that one or more of these factors may play a part in any given case, the varied possibilities accounting for the clinical differences and varying responses to treatment that are observed.

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News and Notes

EDITED BY DR. JOHN HERBERT WAITE

GENERAL NEWS

Von Graefe Served as Soldier.—Three comments have been received, one of them from a reliable source in Germany, denying the truth of the statement in Hasket Derby's article on Graefe, "Der Kerl war nicht einmal ein Soldat." There seems to be no doubt that von Graefe served his Soldatenleben, as attested by the following quotation from the *Hirschberg Geschichte der Augenheilkunde:* "From April 1, 1852, until April 1, 1853, Graefe served his year as physician with the Second Guard Regiment of Foot. Surely, he would not have been called out, since he already looked frail and ill. Because of his outstanding position, very little service was required of him, since the company surgeon was a good fellow and did many things in his stead; moreover, he was given the care of those with eye troubles. When the regular spring parade was held at the Templehof field, he was ordered out with the rest, coming at the last moment, hurrying upon the field with long beard and flowing locks contrary to the regulations of the guard corps, clad in a hastily borrowed and ill-fitting uniform coat. The regimental commander was astounded and shouted: 'Get that man with the frightful beard away from here, and do it so that his majesty does not see him, and so that I never see him again.'"

Fourteenth International Congress of Ophthalmology.—The International Council of Ophthalmology has decided that the Fourteenth International Congress will take place in Madrid early in April, 1933. There will be only two reports and two reporters. The subjects will be determined during the winter.

The International Council now consists of the following: Dr. Treacher Collins, England, Honorary President; Dr. P. Lundsgaard, Denmark, President; Dr. Pflueger, Switzerland, Vice-President; Dr. Leslie Paton, England, Treasurer, and Dr. Marx, Holland, Secretary. Other members include: Dr. P. van de Hoeve, Holland; Dr. Parker, United States; Dr. Wagenmann, Germany; Dr. Oguchi, Japan; Dr. Roselli, Italy; Dr. Wright, Madras; Dr. Coppez, Belgium; Dr. Marquez, Spain, and Dr. Morax, France.

SOCIETY NEWS

Ophthalmological Society of the United Kingdom.—The Fiftieth Annual Congress of the Ophthalmological Society of the United Kingdom will be held in London, April 3, 4 and 5, and the subject for papers and discussion will be "Disorders of Secretion of the Endocrine Glands Associated with Eye Diseases."

Ontario Medical Association.—The association is holding its fiftieth anniversary meeting at Toronto on May 27, 28, 29 and 30, 1930. A program fitting for such an event has been arranged in all branches of medicine.

Chicago Ophthalmological Society.—At the December meeting of the Chicago Ophthalmological Society, Dr. Francisco Payoles, of Madrid, spoke on ophthalmologic activities in the Red Cross Hospital at Madrid.

Cleveland Ophthalmological Club.—The new Cleveland Ophthalmological Club, which held its inaugural meeting in November, has elected Dr. W. E. Bruner chairman, and Dr. M. Paul Motto secretary.

UNIVERSITY NEWS

Prof. Otto Kuffler died suddenly on October 26th, in his fifty-third year. He studied ophthalmology under Axenfeld, Laqueur and Vossius. His scientific contributions were in the fields of serology and bacteriology. His principal work, however, was the editorship of the Springer publications, the *Zentralblatt für die gesamte Ophthalmologie,* and the *Jahresbericht,* which he brought to a point of excellence reflecting the greatest credit on his initiative and industry.

Dr. Francisco Payoles, of Madrid, Spain, on December 18 lectured to the ophthalmologic staff of the Mayo Clinic on "Cataract Operations." With the use of moving pictures, he demonstrated three methods of intracapsular extraction.

Dr. Alois Pampichler, from the Lindner Clinic in Vienna, is temporarily on the staff of the Department of Ophthalmology at the University of Iowa.

It is reported that Dartmouth College has received from Mr. John D. Rockefeller, Jr., a gift of $80,000 to be used for research in physiologic optics.

PERSONAL NEWS

Dr. Jules Gonin Awarded Benoist Foundation Prize.—On November 25, at Bale, the Benoist Foundation awarded its annual prize of 35,000 francs to Dr. Jules Gonin, of Lausanne, in recognition of his treatment for detachment of the retina. Gonin's method consists of accurate localization of the retinal tear, and of attempting to close the tear by means of one or more cautery punctures at its site.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

ON THE CEMENT SUBSTANCE OF THE INTRA-OCULAR MUSCLES AND CHRONIC GLAUCOMA. H. HERBERT, Brit. J. Ophth. **13**:289 (June) and **13**:337 (July) 1929.

According to the author, the work described in this article began primarily as a research into the nature of the cement of the plain muscle of the eye. It is of interest to the ophthalmologist mainly in two of its results: (1) It leads up to and includes the demonstration of a complete glaucoma mechanism; and (2) it throws a good deal of light on the filtration angle and on the ciliary body. The evidence suggests that the cement in the muscles of the human eye differs in quality from that of smooth muscle elsewhere in the body, and from that of the eyes of animals below primates. This raises the question whether it should not be included among recognized hyaline or glass membrane structures of the eye, with a definite rôle beyond that of mere cement. In the ciliary body particularly it appears to be needed as a part of the hyaline system subserving man's highly developed accommodation.

The first part of the article is lengthy, dealing in detail with the histology of the tissues concerned. The author predicates an anatomic basis for the mechanism of glaucoma. He says that many cases of chronic glaucoma are characterized by a combination of three outstanding features:

1. The sinus of the anterior chamber has a thick posterior-inner wall, made up mainly of an extension of ciliary body tissues supporting the iris base instead of iris alone, as in many eyes.

2. The open angle of the chamber has been displaced by pressure of the aqueous, so that it lies abnormally far beyond the level of the scleral furrow and of Schlemm's canal. The position attained may be such as is never seen apart from chronic glaucoma.

3. The meridional bundles of the ciliary muscle fail to reach near to the level of Schemm's canal, and the middle and inner divisions of the muscle are neither particularly broad nor closely packed. There is the defective inward pull of the muscle on the pectinate ligament. And the glaucoma developing in these eyes may be spoken of, not inaptly, as the glaucoma of the peripherally placed and poorly developed ciliary muscle.

The author gives as two strong arguments indicating that the seat of obstruction lies in the pectinate ligament in many cases of chronic glaucoma: (1) clinical evidence of an open filtration angle sometimes persisting for years, as recorded by Troncoso with the gonioscope; (2) Raeder's observations showing that the duration of the influence of physostigmine on the intra-ocular tension in chronic simple glaucoma corresponds to the duration of the influence on the ciliary muscle and not to that of its effect on the pupil. If, then, the site of the obstruction lies in the pectinate ligament the mechanism of the obstruction must

be explained by Henderson's theory of "sclerosis of the cribriform ligament," or anatomically by the author who holds that the Henderson theory lacks support. The article is illustrated.

W. ZENTMAYER.

THE EXAGGERATION OF THE UPPER LIMBUS IN SICKLE-FORM IN CHINESE EYES. ARNOLD PILLAT, Nat. M. J. China **15**:569 (Oct.) 1929.

The purpose of this article is to point out that in Chinese eyes the upper limbus is often overdeveloped, giving the clinical appearance of a white crescent at the upper end of the cornea and making the cornea a marked horizontal ellipse. Two sketches show the exaggeration of the normal limbus and give the exact relation between the width of the limbus and the rest of the cornea. These are interesting as all the measurements of the limbus found in the literature have been made on eyes of the white race. The details of two cases are given. The article closes with a reference to the practical side of these exaggerated limbi, as regards intra-ocular operations, especially those for glaucoma and cataract extraction.

S. H. MCKEE.

Physiology

THE INFLUENCE OF CHANGES IN BLOOD PLASMA ON THE NORMAL INTRA-OCULAR PRESSURE. J. F. DECKER, Arch. f. Augenh. **100-101**: 180, 1929.

Marx has shown that a characteristic curve of changes in the percentage of hemoglobin can be produced in the normal person by drinking a large quantity of water. At the same time the elimination and specific gravity of the urine show characteristic changes. The character of the curve depends on the condition of the water balance before the ingestion of the fluid. In order to study the changes in intra-ocular pressure that occur after the ingestion of fluid, the author studied fifteen normal persons of varying ages and of both sexes. For two days the subjects were given a constant diet with the addition of 1,000 Gm. per fluid daily. On the third morning a liter of water was ingested as rapidly as possible and thereafter estimations of hemoglobin, collection of urine, and determinations of intra-ocular pressure were made at regular intervals. In spite of the fact that the hemoglobin and the urine elimination followed the characteristic curve of Marx, no change could be shown in the normal intra-ocular pressure.

An intravenous injection of 10 cc. of 10 per cent salt solution, or from 40 to 60 cc. of a 20 per cent solution of dextrose was next given to a series of subjects; the hemoglobin was estimated and intra-ocular pressure determined in the usual manner. The hemoglobin fell as the blood plasma became diluted but no definite changes could be shown in the intra-ocular pressure. It appears that the eye can maintain a normal intra-ocular pressure in spite of relatively large changes in the volume of blood plasma. This was further proved by an experiment performed on Marx himself as follows:

His normal blood plasma volume was about 4,150 cc. After four days of a strict dry diet, the volume of plasma had diminished to 2,280 cc., with a loss in weight of 3.5 Kg. Following this, a four day period of high ingestion of water was begun. He received 2,500 Gm. of water

and 1,500 Gm. of 1 per cent salt solution. At the end of this period, his plasma volume had risen to 6,550 cc. In spite of these marked changes in volume of plasma, no change could be found on repeated determinations of his ocular tension.

Similarly, hot baths produced definite changes in the hemoglobin concentration. A slight fall of intra-ocular pressure occurred, probably due to the fall in the blood pressure which was found in these subjects.

F. H. ADLER.

Methods of Examination

THE BLIND SPOT. H. S. GRADLE and S. J. MEYER, Am. J. Ophth. 12:802 (Oct.) 1929.

The history, anatomy, pathologic significance of the blind spot, indications for outlining it, size and location and methods of measurement are discussed. Among the latter, an ingenious "confrontation test" is given. This is recommended particularly for neurologic patients whose fixation is difficult to control. It consists of a meter string with a white headed pin tied in the center. One end of this string is held by the patient on a level with his right eye while the observer holds the other end and compares his blind spot with the patient's by carrying the pin temporally into the field of the blind spot. This article does not lend itself to abstracting and must be read to be appreciated.

W. S. REESE.

INTENSITY OF ILLUMINATION AND OTHER FACTORS INFLUENCING THE SENSITIVITY OF THE RADIAL TEST FOR ASTIGMATISM. C. E. FERREE and G. RAND, Am. J. Ophth. 12:809 (Oct.) 1929.

This paper deals with a method of testing for astigmatism by playing on intensity of illumination. Charts of radial line type are used with a very high intensity of illumination. The following conditions must be met: 1. The difference in coefficient of reflection between the lines and their background should be as great as possible. 2. The illuminating light should be high intensity. 3. The lines should be narrow. 4. The incident light should be white. 5. There should be very low general illumination. Several charts and tables are given. The authors claim to be able to detect an astigmatism of less than 0.12 diopter.

W. S. REESE.

Experimental Pathology

EXPERIMENTAL CONTRIBUTION TO THE MECHANISM OF CICATRIZATION IN ANTERIOR SCLERECTOMY. L. MAGGIORE, Ann. di ottal. e clin. ocul. 57:579 (Aug.-Sept.) 1929.

Previous works on the subject indicate that healing of the sclerectomy wound by firm connective tissue is prevented only when uveal tissue is included in the wound, either deliberately, as in iridencleisis, or accidentally by iris prolapse during iridectomy or penetrating wound. The author's description of five human eyes with sclerectomy or penetrating wounds bears out this previous evidence. One case showed especially well the effect of included uveal tissue. It was trephined above and

three months later also below. The first wound was free of uveal tissue, and closed firmly by direct growth of scleral fibers. The second wound was partly filled by a prolapse of ciliary body, and here the scleral wound remained open. Sclerectomies were performed on rabbits in various ways to study the mechanism of cicatrization. Where the uveal tract was well separated so that none prolapsed into the wound, the largest sclerectomy openings healed firmly. Iris inclusion was made in another series by turning a flap of iris out after two meridional incisions, the pigment epithelium being turned outward so as to come in contact with the corneal edge of the wound. This effectively prevented cicatrization from the sclera. Equally effective was the inclusion of free fragments of iris which had been removed at iridectomy from the same eye or the opposite eye. Uveal tissue from albino rabbits was not as effective as that containing pigment epithelium. In another series of experiments, a piece of conjunctiva was included between the lips of the wound. Cicatrization was impeded, but the connective tissue which replaced the conjunctiva was too firm to allow any filtration. A piece of the superior rectus was placed between the wound in several other animals. It was well tolerated and prevented the sclera from cicatrizing. The tissue was apparently much more favorable for filtration than ordinary connective tissue. Attempts to produce free filtration by inclusion of a small glass tube and a piece of mica were unsuccessful, the tube becoming closed with fibrin, and the mica being surrounded by dense connective tissue. (Ten photomicrographs.)

S. R. GIFFORD.

CATARACT PRODUCED BY SODIUM CHLORIDE. E. PANICO, Ann. di ottal. e clin. ocul. **57**:613 (Aug.-Sept.) 1929.

Previous work on the production of cataract by the use of various salts is reviewed. Solutions of sodium chloride, when instilled in the conjunctival sac or injected intravenously in sufficient amounts, have been shown to produce opacities of the lens. Manca and Ovio found that this depended entirely on the hypertonicity of the solution and the consequent extraction of water from the lens with resulting loss in weight. They found that an isotonic solution for beef lens, which caused no change in weight, was 1.2 per cent sodium chloride. The author tried the effect of injecting solutions into the anterior chamber and the vitreous chamber of rabbits. The resulting changes in the lens were studied with the slit-lamp and in sections of iris removed at various ages. A few drops of a saturated solution produced an intense reaction with complete opacification of the lens. After seventy hours this had cleared up except for a localized opacity under the anterior capsule of the lens, which persisted after two months. With a 3 per cent solution, the opacities of the lens were limited to stripes in the anterior cortex suggesting the suture lines. These had cleared up in four hours. With 2 and 1 per cent solutions, the changes were much slighter, disappearing with the latter solution after forty minutes. When injections were made into the vitreous, the same changes were observed, but were confined chiefly to the posterior cortex. The author attributes the changes entirely to the hypertonicity of the solution used, the extraction of water from the lens causing opacity which usually disappears as soon as the

water is replaced by absorption. When the loss of water occurs too suddenly and persists too long, as with the stronger salt solutions, the lens fibers are permanently damaged. (Bibliography.)

S. R. GIFFORD.

EXPERIMENTAL OCULAR TUBERCULOSIS. B. COURTIS, Arch. de Oftalm. de Buenos Aires **4**:75 (Feb.) 1929.

The author used well nourished rabbits in good physical condition without any eye lesions. He describes in detail the technic employed, the results obtained, and the histopathology of the ocular lesions produced. He gives the clinical history of four cases of human ocular tuberculosis. He considers ocular tuberculosis secondary in nature, the eye infection taking place through the blood stream.

His conclusions are:

1. The tuberculosis inoculated into the eye of the rabbit reproduces the characteristics of the ocular tuberculosis in man.

2. The clinical features, the form of evolution and the localization of the experimental ocular tuberculosis in the rabbit confirm the secondary nature and the endogenous origin of ocular tuberculosis.

3. The order of frequency in the lesions produced was: iris and ciliary body, sclerotic, cornea, deeper membranes of the eye and lens.

4. The surest site for a successful inoculation is the anterior chamber.

5. This constitutes a sure and excellent means of diagnosis.

The paper is illustrated by numerous colored plates and photomicrographs and is accompanied by a complete bibliography.

C. E. FINLAY.

THE DEVELOPMENT OF THE ZONES OF DISCONTINUITY IN THE LENS. H. GOLDMANN, Arch. f. Ophth. **122**:198 (May) 1929.

The author studied the experimental cataract caused by naphthalin, tetany and x-rays in dogs, rats and rabbits. He found that zones of discontinuity always appeared near the anterior surface of the lens in intimate connection with the toxic lesions. The cataract which is produced by x-rays in rats may be latent for a long time. X-rays influence the epithelium of the lens, and the cataract begins to show much later. Sometimes the lens fiber becomes opaque when it is already deeply embedded in the cortex of the lens, although the corresponding epithelium cell of the lens was injured at the time when it just started to grow. The easiest way to explain the zones of discontinuity in the normal lens is to assume that they represent periods during which the growth of the lens fibers was impeded or retarded. As the refractive index of the lens fibers increases with age, a layer of fibers which get old without much growth must form a whole zone with a different refractive index or, in other words, a zone of discontinuity.

P. C. KRONFELD.

Conjunctiva

LATER NOTES ON THE TREATMENT OF VERNAL CATARRH WITH EPINEPHRINE. G. TESSIER, Ann. di. ottal. e clin. ocul. **57**:631 (Aug.-Sept.) 1929.

The author refers to his previous favorable reports on the use of epinephrine by subconjunctival injection. Since his last report in 1927,

he reports eighteen new cases in which this treatment was used and gives a report on several in which treatment was begun before that date. From eight to fourteen injections of from 0.12 to 0.33 cc. of 1:1,000 epinephrine were given, the intervals between injections being from three to four days. Of the eighteen new cases, cure was reported in ten and a disappearance of limbar and improvement of tarsal lesions in four. There was improvement in the two other cases and in two, both of the tarsal form, the treatment had no affect. In two previous cases which had shown recurrences, relief was obtained by a further course of injections. The most favorable cases were those of the limbar type. All cases showed an eosinophilia in the secretion and one or more signs of glandular imbalance. The author believes the treatment with epinephine to be the one of choice, the use of other glandular extracts, such as testicular extract, being of probable value in some cases with signs pointing to deficiency of such secretions. (Bibliography.)

S. R. GIFFORD.

VERNAL CONJUNCTIVITIS TREATED WITH LACTIC ACID. S. TRONTSCHEFF, Klin. Monatsbl. f. Augenh. **83**:48 (July) 1929.

The author used lactic acid for the first time in vernal conjunctivitis. He treated nineteen patients, the majority of whom were less than 26 years of age, and obtained a perfect cure in twelve. After anesthesizing the conjunctival sac with a 5 per cent solution of cocaine to which epinephrine is added, Jaeger's horn plate is inserted below the inverted upper lid, and a 10 per cent solution of lactic acid is applied with a cotton-tipped probe. The surface is neutralized with physiologic sodium chloride or boric solution or mercury oxycyanate after one or two minutes. The same process is repeated for the lower lid which is usually less involved, and for the conjunctiva of the globe. The cornea must be carefully protected. The eyes will be free from pain after this procedure, which will stop the photophobia. No bandage is needed. Itching of the lids and secretion will cease about three days after the application. The treatment is finished within an average of two or three weeks. Solutions of from 30 to 40 per cent are used in some cases, causing pain about equivalent to that felt after application of a 2 per cent solution of silver nitrate. The treatment with lactic acid is to be repeated in some instances and always after curetting of proliferations. Involved portions of the conjunctiva of the globe may have to be excised. The wound will heal without complications within a week.

Discoloration of the portions treated with lactic acid has not been observed. A macula of the cornea, incidentally caused by a drop of a concentrated solution, disappeared after a short time. General treatment, in the author's opinion, is useless, because the vernal conjunctivitis is not due to conditions of the organism or to toxins.

K. L. STOLL.

Cornea and Sclera

THE ETIOLOGY OF KERATOCONUS. BRUNO FLEISCHER, Arch. f. Augenh. **100-101**:247, 1929.

Fleischer reports the case of a young girl who developed a keratoconus in one eye after the opposite eye had been injured. The corneal curva-

tures of the uninjured eye were not measured at the time of the first examination, but the author concludes that the visual acuity which was almost normal at that time precludes the possibility that a keratoconus was present at that time.

About one year after the injury, the patient was examined again, and a well developed keratoconus was discovered. A review of the various theories of the etiology of keratoconus is given, and the author concludes that this condition may arise from a number of causes, all of which have as a common factor weakness of the central area of the cornea.

F. H. ADLER.

FAMILIAL NODULAR KERATITIS. R. GUTZEIT, Ztschr. f. Augenh. **68:** 349, 1929.

From his study of a small group, Gutzeit concludes that there are several variations in the lesion which is classed as familial nodular keratitis. He substantiates his opinion by reviewing several case reports found in the literature. Nodular keratitis appears early in childhood. The lesion, as a rule, is not accompanied by any visible congestion. It is progressive in nature but does not lead to complete blindness.

A. M. YUDKIN.

Lens

HEREDITARY, FAMILIAL AND MYOPATHIC CATARACT. F. TERRIEN, P. SAINTON and P. VEIL, Arch. d'opht. **46:**193 (April) 1929.

The authors describe in detail the case of a man, aged 37, who had gradually become blind over a period of eight years. In addition to the cataracts he had a marked atrophic muscular condition which was more advanced in the lower extremities. The patient's paternal grandmother, his father and a paternal aunt had had cataract. None of them had any symptoms of myopathy.

The patient presented the triad of symptoms described by Foix and H. LaGrange: (1) a distal type of myopathy, (2) a myotonic reaction with abolition of the tendinous reflexes and (3) a special dystrophy. They suggest that a fourth symptom, bilateral cataract of a specific type, can properly be added. The cataract is of the type already described and pictured by Vogt and Lussi. They do not think, however, that the discovery of such a cataract is pathognomonic of myotonic dystrophy. Moreover, the latter condition should not be diagnosed from the appearance of the cataract alone. They support the view that this form of cataract is acquired, although they admit that it is familial and hereditary. In their patient, it appeared to be acquired; the myopathy, however, was not. The coexistence of the two conditions is rare. They call attention to the possibility of a common endocrine disorder as the underlying causative factor in both conditions, but they doubt that this is the case. The lesions found in the nervous system have been agreed on by L. Nicolesco, M. Nicolesco and Foix to be those of senescence. They raise the question as to whether the cataract is not of a similar nature. It would be a common observation in both conditions.

S. B. MARLOW.

PRACTICAL EXPERIENCES WITH CATARACT OPERATIONS ON OLD PEOPLE. NICHOLAS BLATT, Orvosi szemle, April, 1929.

This series of operations for cataract includes forty-six patients, all over 80 years of age; eleven were over 90, and one woman was 103 years old. All were operated on in private houses. Graefe's extraction with large corneal and conjunctival flaps and with iridectomy proved to be the most appropriate. Operations done on such old persons need particularly careful preparation, performance and after-care on account of the patients' great sensibility and reduced resistance. We ought, therefore, to proceed rather conservatively and, if swerving from Graefe's classic operation, to do so to the smallest possible extent. Other methods, such as extraction without iridetcomy, peripheral iridectomy, Smith's intracapsular extraction and linear extraction, give no good results in old people and are often too dangerous. Compensated cardiovalvular defects, chronic bronchitis, emphysema of the lungs, chronic interstitial nephritis, diabetes mellitus, chronic constipation and gastro-intestinal disturbances, only too often encountered in old people, are not contraindications to operation. Medical treatment should, of course, be previously undergone. Also, most of the psychic alterations do not oppose the performance of the operation. The condition of the lacrimal passages is, of course, of great consequence. Prognosis regarding vision is only dependent on the perception of light by the cataractous eye, but not always on the behavior of the pupillary reaction, because the latter may still be present in spite of severe amblyopia, and vice versa. Operation should be done as soon as patients have become unable to find their way about. It is not necessary to await maturation of the cataract.

Five per cent cocaine drops should be given for fifteen minutes, and atropine only on the second day, if necessary; if there is severe conjunctival discharge, 0.3 per cent silver nitrate should be administered. Postoperative rupture of the wound is for the most part not due to injuries proceeding from fidgety patients, but to swelling remnants of the crystalline lens, to iris prolapse, blood clots remaining in the wound during operation and counteracting normal and smooth union of the margins of the wound. Extraction of eventual secondary cataracts is more beneficial than discission and less dangerous, but it should not be done before from two to three months after the first operation.

AUTHOR'S ABSTRACT.

Glaucoma

CLINICAL FEATURES IN CASES OF GLAUCOMA. C. ESPÍLDORA LUQUE, Arch de oftal. hispano-am. **30**:341 (July) 1929.

In this paper the author studies patients in different age groups from a general point of view from the local condition of the eye.

Glaucoma is to be found in youth, although much less frequently, only 25 of the 300 cases occurring in patients less than 40. Study of these is especially interesting as possibly suggesting an etiologic factor, which later may be masked by other features. In nineteen of these twenty-five cases syphilis was found to be present, in only four of which there was some element of doubt. Another almost constant feature was the presence of lesions of the cardiovascular apparatus (aortitis, valvular

insufficiencies, arterial hypertension). In one of the cases, that of a girl, aged 15, a typical acute glaucoma yielded to myotics and to specific treatment without operative intervention. In one of the four patients with doubtful syphilitic antecedents, there was arterial hypertension, in one aortitis and in two (women) in whom no general lesions were found there were histories of multiple abortions. Of the six cases free from any suggestion of syphilis, two acute attacks occurred during the final stages of severe attacks of grip; in three of the four remaining cases, no general lesion could be found, and in one there was a chronic myocarditis with rupture of the vascular equilibrium.

Among mature and presenile adults in whom the majority of cases of glaucoma occur (161 of 300), the author finds syphilis and vascular disease (arteriosclerosis) most frequently accompanying glaucoma. Syphilis does not appear so frequently as in youths (77 of the 161—59 with a clear history and 18 with a doubtful history). Among these there was no organic lesion in ten, syphilis being discovered by a positive Wassermann reaction. More frequently, however, a positive Wassermann reaction is accompanied by some vascular lesion, especially an aortitis with or without valvular involvement, arterial hypertension being at times combined with arterial hypertension. Another group with a negative Wassermann reaction presented aortitis, Argyll Robertson pupils and other signs of syphilis.

The vascular lesions in these patients with glaucoma can be classified in three groups: (1) chronic aortitis, without any apparent etiology; (2) essential arterial hypertension, and (3) both aortitis and hypertension.

These were in much inferior proportion to syphilis—from 22.7 to 48 per cent. In most cases it is impossible to determine the etiology of these vascular lesions; there were, however, thirty-six cases in mature and presenile patients with arteriosclerosis in whom syphilis was evident. In some cases the lesions were limited to the aorta and in others they extended throughout the vascular system. Some of the patients had a normal arterial tension, but most frequently there was some arterial hypertension. In some cases, the condition was complicated with renal dyspnea. In the senile patients vascular lesions, mostly arteriosclerotic were observed frequently, while syphilis was noted less frequently.

C. E. FINLAY.

VASCULAR NEVUS AND GLAUCOMA. K. KRAUSE, Ztschr. f. Augenh. **68**:244 (July) 1929.

A survey of the literature showed that the twenty-seven cases reported could be divided as follows: (1) those cases in which there was an increase of intra-ocular pressure in middle life in the form of glaucoma simplex and (2) those in which hydrophthalmus accompanied the skin lesion from early childhood. Krause carefully reviews the opinions of many investigators and also presents two cases which he places in their respective groups. He concludes that the ocular disturbance in the first group is due to derangement of the endocrine secretion following growth stimulation. He assumes that in the second group the hydrophthalmus is due to an abnormality of Schlemn's canal and engorgement of the choroidal vessels. The clonic spasm which frequently accompanies this group is probably produced by a hemangioma of the brain.

A. M. YUDKIN.

A Case of Primary Glaucoma with Morning Hypotony. Diego Diaz Dominguez, Arch. de oftal. hispano-am. 30:491 (Aug.) 1929.

After calling attention to the difficulty in establishing a diagnosis in certain cases, with normal tension at the time of examination, the author refers to the investigations of Kölner, Thiel and Langenham, recently elaborated by Löhlein, establishing variations in intra-ocular pressure at different hours, with formation of a tension curve, which in glaucoma is characterized by a morning elevation and a fall toward evening.

He next reports a case of primary glaucoma with an inversion of the daily oscillation, the highest tension being noted in the morning, the lowest in the evening.

He refers to cases of inversion reported by Raeder, not accepting his explanation on account of it not being in accord with modern developments in our knowledge of the nature of intra-ocular fluids (Duke-Elder and others).

C. E. Finlay.

Retina; Optic Nerve

Sclerosis of the Retinal Vessels. N. Pines, Brit. J. Ophth. 13:97, March; 161, April; 225, May, 1929.

The author's conclusion is that in spite of the lack of direct anatomic evidence, the vessels in the normal eye run on the disk and on the retina through channels of perivascularis. The disk is covered by a transparent and strong membrane of mesodermic origin. The vessels on the disk are under a pressure in the sagittal and horizontal diameters which is greater than that on the retina.

When they become sclerotic they first lose their translucency, then change their color, and "dot" their light reflex; then the sclerotic perivascularis appears, first as white lines at the crossing points, and later on it takes a leading part in arteriovenous compression. This thickened perivascularis plays an important part in every pathologic vascular process in the sclerotic eye, but its action is chiefly local, affecting separate parts of the vessel, especially at crossing points. Essential hyperpiesis greatly increases and aggravates all the sclerotic signs of the retinal vessels. It is probable that there is a predilection for the retinal vessels, and that these are affected earlier and more severely than other parts of the vascular tree except the cerebral vessels and perhaps those of the kidneys.

Syphilis per se does not especially attack the retinal vessels, but if arterial hypertension is also present, the sclerotic changes are well marked and advanced, as in similar cases of essential hyperpiesis.

Atheroma of the heart or large vessels, even in a severe form, with normal blood pressure, does not produce any special changes in the retinal vessels; on the other hand, a severe atheroma of the retinal vessels and even of the cerebral ones, may spare the heart and the aorta. Essential hyperpiesis, when added to atheroma, produces marked and advanced retinal changes which depend chiefly on the degree of arterial hypertension.

Even mild diabetes attacks the retinal vessels, but most probably to the same degree as the vessels are affected in the other parts of the body. Constant, frequently repeated, and careful observation of the

urine, blood pressure and retina is necessary in every case of diabetes, whether the treatment is purely dietetic or the administration of insulin; aggravation of the retinal changes, hemorrhages, and especially edema are often combined with rise of blood pressure and lowering of the specific gravity of the urine.

W. ZENTMAYER.

OPTIC NEURITIS AND DENTAL SEPSIS. E. TEMPLE SMITH, Brit. M. J. 2:99 (July 20) 1929.

A man, aged 35, whose vision in the left eye was reduced to 6/12, with blurred nerve-head and the field examination showing enlargement of blind spot, and two small scotomas, had two infected upper bicuspid teeth. After extraction, complete recovery resulted in seven weeks. The writer praises the Elliot scotometer in the detection of central field defects.

A. KNAPP.

THE CAUSES OF RETROBULBAR NEURITIS. R. SCHEERER, Klin. Monatsbl. f. Augenh. 83:164 (Aug.-Sept.) 1929.

Acute and chronic retrobulbar neuritis was discovered in 203 of 64,200 patients examined at the University Eye Clinic of Tuebingen during the seven years ending 1928. After the elimination of all other causes, 134 cases were due to multiple sclerosis and 3 to disease of the accessory nasal cavities. Unilateral and bilateral neuritis occurred equally as often with both sexes equally represented. Only 5 of the 134 patients were temporarily blind for periods of from six to fourteen days. All regained full vision with two exceptions; in one case the vision was 5/12 and in another there was permanent amaurosis. Besides the three cases of certain rhinogenous origin, three others were doubtful, for here after opening the cavities, the mucous lining was found to be normal. Twenty other cases, doubtful at first, proved later to be of divers origin, but not due to nasal disease. Prompted by these observations and disagreeing with other writers, the author considers retrobulbar neuritis of rhinogenous origin rare and due only to special anatomic relations between the optic nerve and the cavities. Geographic and racial influences may have to be considered.

Good results were obtained in retrobulbar neuritis due to multiple sclerosis by the administration of 0.15 cc. of neoarsphenamine, weekly, besides the general treatment. Some improvement of the vision was frequently observed on the day following the injection.

K. L. STOLL.

OBSERVATIONS ON THE CLINICAL PICTURE OF RETINITIS (COATS), RETINAL DEGENERATION WITH MULTIPLE ANEURYSMS (TH. LEBER) AND ANGIOMATOSIS RETINAE (CZERMAK-VON HIPPEL). PAUL JUNIUS, Ztschr. f. Augenh. 68:207 (July) 1929.

It is interesting that all three retinal diseases occur most frequently in young people. Von Hippel's disease is the only one in which definite congenital manifestations—familial in nature—have been noted. Lindau designated this disturbance as hemangioblastoma and noted that it makes its appearance most frequently in the third decade. Junius noted that

this latter disease presents other manifestations of the same "blood tumor" disturbance in earlier periods of life in such areas as the medulla oblongata, kidneys, suprarenals and pancreas. It is significant that all three diseases involve the small retinal vessels, particularly the small arterioles. There is an increase of capillarization from sausage-like swellings to that of aneurysm with variations of the caliber of the vessels to tumor forms, as seen in von Hippel's disease. It is difficult to determine whether the vascular changes are primary or secondary.

A. M. YUDKIN.

FAMILIAL ANGIOMATOSIS RETINAE ET CEREBELLI (LINDAU'S DISEASE). H. U. MÖLLER, Acta ophth. **7**:244, 1929.

From a survey of the literature, Möller finds that there is an association of angiomatosis retinae, von Hippel's disease, with a similar disease of the central nervous system. Several authors, particularly Lindau, believe that both conditions are due to a congenital anlage. It is difficult to determine which disturbance manifests itself first. The disease may be present only in the cerebellar tissue, yet there is a possibility that a retinal angioma is also present simultaneously in all cases, but of microscopic size. Most of the cases reported disclose a familial tendency. The author suggests that the diagnosis of "Lindau's disease" should be made only when the following factors can be demonstrated: cerebral tumor plus familial incidence, or retinal angiomatosis plus cerebral tumor, with or without familial tendency.

A. M. YUDKIN.

Society Transactions

EDITED BY DR. GEORGE S. DERBY

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

London, England, Oct. 11, 1929

MR. CYRIL H. WALKER, *President, in the Chair*

CYSTIC ADENOMA OF THE CONJUNCTIVA. Presented by MR. COLE MARSHALL.

Mr. Marshall said that the adenoma was as freely movable as a limpet; in fact the patient sought examination because it got over the pupil of his eye. Under the idea that it was angioma Mr. Marshall removed it freely and sectioned it, and found it to be a cystic adenoma.

A CASE OF PELLET IN THE VITREOUS. Presented by MR. HUMPHRY NEAME.

A man had received a gunshot wound in the eye. Mr. Neame felt that the eye would have to be removed, as it was in a bad condition and looking angry. However, as it showed signs of improving after a fortnight he allowed it to remain, and the pellet was still visible.

PERSISTENT PUPILLARY MEMBRANE. Presented by MR. J. D. CARDELL.

A boy was admitted to the hospital in February, having received a blow in the left eye while wearing his glasses. The glasses were shattered, and a wound of the cornea and prolapse of the iris resulted. The prolapse was removed and the eye did well. A few days afterward he discovered, with the slit-lamp, a structure in the pupil resembling a cilium that was implanted in the iris, and a similar structure on a parallel but deeper plane; this also resembled a cilium, though it was improbable that two cilia had become implanted in this way. There had been no inflammation in the eye, and the condition had remained stationary.

DISCUSSION

MR. ERNEST CLARKE: I had a case in which a cilium was apparently growing in the iris. I removed what I thought was the cilium. The eye became normal, but I think the cilium was still there.

MR. HUMPHRY NEAME: I think that two cilia entered at the same time.

BILATERAL SYMMETRICAL HYPERPLASIA OF THE LOWER CONJUNCTIVAL FOLD. Presented by MR. O. G. MORGAN.

The patient was a surgeon who had been in practice in the West Indies. His eyes had been red off and on for some months, but the condition had not worried him beyond causing irritation. In the lower conjunctival fold was a thick, hard hyperplasia, while on the caruncle were papillary elevations. There were no enlarged glands, and the Wassermann reaction was negative.

The diagnosis, according to Mr. Morgan, lay between spring catarrh and tubercle. No eosinophils occurred in the smear examined. After a portion had been removed, he intended to have a section made.

DISCUSSION

COL. A. S. J. LISTER: I think that the condition is spring catarrh, though it is not in accord with the types seen in England. In hot climates, where cold does not alternate with heat, a continuous irritation occurs that is not seen in this country.

CHOROIDITIS WITH INFILTRATION OF THE VITREOUS. Presented by DR. SOURASKY.

Instead of floating opacities, the whole vitreous seemed to be replaced by a dense mass of shrinking tissue. All examinations gave negative results, and the question arose whether the dense tissue could be due to the presence of a foreign body. The results of the roentgen examination were negative.

SCHNEIDER CATARACT. Presented by DR. SOURASKY.

The patient was a young woman whose work was connected with induction coils. There was considerable keratitis punctata that had been increasing. Observers had failed to detect a foreign body; the vitreous and fundus seemed normal. The foreign body was nonmagnetic and no help was obtained from the slit-lamp.

INTRA-OCULAR FOREIGN BODY. Presented by MISS DOLLAR.

A woman, aged 33, had severe diabetes. In this case the capsule was replaced by a dense layer.

DISCUSSION

MR. B. CRIDLAND: I recall a case in which a piece of copper from a percussion cap entered the eye, but without destroying the sight. I felt that I could easily remove it and open the globe. I made what I thought were successful attempts at removal with sclerotomy forceps, but failed. No keratitis punctata developed, and I do not know the subsequent history of the case.

THE CHEIROSCOPE. Demonstrated by MR. E. E. MADDOX.

Mr. Maddox said that squint treatment is of good value in selected cases. He puts the hand into service in the process of educating the eye. The instrument he demonstrated is to be used when the squinting eye needs retraining. There are four ways of using it: (1) the covered mirror, (2) the absent mirror, (3) the oblique mirror and (4) the vertical mirror. One eye sees the picture and the other sees the pencil that draws it. He regards the drawing as important. Children grow tired of looking at the same picture over and over again, without mental concentration, and little good is effected. Full correction of refraction is necessary. Bifocal glasses lessen the accommodation for reading. There should be a long continued occlusion of the fixing eye. It is difficult to know when to operate in a given case. When the nonoperative measures are unsucessful, or require a long time, so as to jeopardize eventfully the cooperation of the brain and eye, operation is desirable as soon as its inevitability is apparent, regardless of the youth of the patient. Mr. Maddox has even done the operation on a boy 18 months of age, and the eye has been in good condition ever since.

DISCUSSION

MR. ERNEST CLARKE: I should like to ask about the use of the bifocal glasses. As to the methods of occluding the eye, putting a clip over the eye is useless. I put as a minimum requirement the wearing of a shield during mealtime, and if the patient can wear it for an hour afterward, without protest, so much the better.

MR. B. CRIDLAND: My best results came from occluding the eye all day long. My method is to stick the lids together with rubber plaster. Such a procedure does not allow of peeping round the edges, and in a week's time the vision can be improved from 6/60 to 6/6.

MR. O. G. MORGAN: I think that it is better to straighten the eyes and start fusion immediately. I would operate first and then use the cheiroscope.

MR. E. E. MADDOX: I only use atropine when the parents' objection to an occluder seemed too great to be overcome. Atropine is only a partial agent, as it only partly obliterates the noxious influence of the brain on the squinting eye. I agree with Mr. Cridland that occlusion cannot be too constant. In one child the eye was completely occluded for three months, and his vision improved from 6/60 to 6/12 by that measure only. There is need for some uniformity of advice on this subject, for if parents go to one surgeon and receive from him a severe sentence, and go to another surgeon and receive a light sentence, they naturally prefer the latter surgeon. The best plan is never to have both eyes open at the same time during occlusion, because when both are uncovered the good eye interferes with the other. One cannot tell whether one will get fusion until it occurs; all that one can do is to aim at it. There are two kinds of squint, both dating from birth. One is essential squint, and a child with such a condition cannot be educated, as one cannot train what is not present. A child with the other kind of squint, which also dates from birth, might be successfully trained. I agree with Mr. Morgan that the squint training is much more advantageous after the eyes have been operated on. Sometimes one has to concede too much to parents in the matter of the use of atropine.

SOME OCULAR COMPLICATIONS OF NASAL SEPSIS. Presented by MISS ROSA FORD.

Miss Ford read a paper dealing with the causal connection between sinus disease and ocular manifestations, and advocated a closer cooperation between the rhinologist and the ophthalmologist. She thinks that there is need for some more conclusive rhinological tests of the presence of a latent sinusitis. The paper was briefly discussed by several members.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Annual meeting, Oct. 21, 1929

VISUAL FIELD STUDIES. DR. WILLIAM D. ROWLAND and ALLAN WINTER ROWE, PH.D.

Routine studies of the visual fields were made on a series of 1,900 unselected patients seen in the course of a general diagnostic service.

Of these, 102 had visual fields which showed marked concentric contraction for both form and color. These patients were carefully tested by all the routine office examinations, and the usual recognized causes of concentric contraction of the visual fields were ruled out.

The authors feel, therefore, that this study demonstrates that a number of other disease conditions may produce this condition. Endocrine disorders are first considered, and of these, disorders of the pituitary, not necessarily associated with hyperplasia of the gland, are of chief importance. Disturbance of the thyroid or the gonads may also be of minor importance. Lesions affecting the central nervous system in syphilis, tuberculosis and hepatic dysfunctions are also mentioned as probable causal agents.

Finally, tests of kidney function seem to show that in these cases with characteristic eye changes, there was evidence of a functional disturbance of the kidney.

THE FREQUENCY OF SYMPATHETIC OPHTHALMIA. DR. GEORGIANA DVORAK-THEOBALD.

The author had gone over the records of the Illinois Eye and Ear Infirmary and had found during a period of twenty years, ten cases of sympathetic ophthalmia among from 216 to 592 case records. Of these cases of sympathetic ophthalmia, four occurred during the first decade and only one case for each of the following decades. However, the number of perforating injuries was greatest during the first decade and dropped thereafter in proportion to the number of cases of sympathetic ophthalmia.

Statistics showed that sympathetic ophthalmia was much less frequent than the enucleations which were done for the disease or in order to prevent it. Since we had no certain means of deciding in an early stage whether an inflammation was sympathetic ophthalmia or a less harmful inflammation, these enucleations were justified. The diagnosis could be made only histologically. The histologic details of several cases were given with photomicrographs.

CONSERVATION OF THE LACRIMAL SAC. DR. F. N. KNAPP.

The author gave the technic of the various operations devised for removal of the lacrimal sac with maintenance of intranasal drainage and described a modification of the Dupuy-Dutemps operation.

SOME ORIGINAL EXPERIMENTS WITH THE O'CONNOR MUSCLE SHORTENING OPERATION. DR. JOSEPH L. MCCOOL.

The author described the O'Connor muscle shortening operation and gave a table of the shortening produced by using different sized strands of dermal and by varying the width of the lateral strips.

When lateral strips of tendon measuring from 2 to 2.5 mm. in width were shortened with fine dermal, each strand would effect a shortening of 0.55 mm. With medium dermal, the shortening per strand was 1 mm. When lateral strips of tendon measuring 3 mm. in width were shortened with fine dermal, each strand would effect a shortening of 0.66 mm. With medium dermal, the shortening per strand was 1 mm.

REMOVAL OF A NO. 8 LEAD SHOT FROM THE VITREOUS WITH 20/20 OR 6/6 VISION IN ONE CASE. DR. GEORGE H. CROSS.

Using especially constructed wire loop forceps and a double fluoroscope, the author successfully removed lead shot from the eyes of a series of patients. In one case, vision of 20/20 was secured in an eye which would have been lost had the shot not been removed.

DESTRUCTIVE PURULENT OPHTHALMIA ACCOMPANYING AN ERUPTIVE FEVER WITH STOMATITIS. DR. JOHN M. WHEELER.

Two cases were reported of an eruptive fever accompanied by a purulent conjunctivitis. In each case, the skin eruption showed certain identical characteristics, consisting of oval, dark reddish macules separated by normal layers of skin. In a few days these became firm, brownish-purple papules without subjective symptoms. The mouth and lips were especially affected. In one case, the purulent conjunctivitis progressed to panophthalmitis and total loss of vision. In the other case, it responded to treatment but left a severe corneal scar. The true diagnosis of this condition must be left open. The symptoms strongly suggest an infectious disease of unknown etiology, and the author believes that this condition deserves to be considered a definite clinical entity.

RECURRENT HEMORRHAGES INTO THE RETINA AND VITREOUS; CALCIUM DEFICIENCY AS POSSIBLE CAUSE. DR. CHARLES A. YOUNG.

The author detailed the possible etiology of recurrent hemorrhages into the retina and vitreous in adolescents. Of the three cases reported, the first showed a considerable deficiency in blood calcium, and the administration of calcium seemed to be beneficial. The author advised that calcium deficiency should, therefore, be kept in mind as a possible cause of the disease.

RECIPROCAL INNERVATION OF THE EXTRA-OCULAR MUSCLES. DR. FRANCIS HEED ADLER.

Two cases were reported of paralysis of the third nerves in which the eye could be adducted from the position of rest when the fellow eye was strongly abducted. The internal rectus muscle being paralyzed, the adduction of this eye could be accomplished only by a relaxation of the tone of the external rectus in association with an increase in tone of the external rectus of the opposite eye. These cases demonstrated the fact that reciprocal innervation does exist in the extra-ocular muscles of man.

POST-TRAUMATIC OCULAR TUBERCULOSIS. DR. NELSON MILES BLACK and DR. F. HERBERT HAESSLER.

Two cases were reported, one following extraction of a cataract, the other following trauma to one eye in which a tuberculous iridocyclitis occurred.

ACCESSORY EFFECTS OF THE CORRECTING GLASS BY ITS POSITION BEFORE THE AMETROPIC EYE. DR. ALFRED COWAN.

The ideal position of a correcting lens before an ametropic eye is when its pole coincides with the visual line, its second principal point

coincides with the anterior principal focus of the eye and its posterior focus coincides with the punctum remotum of the eye.

Any variation in position introduces a more or less serious error. In the adjustment of glasses, the geometric center of the lens should coincide with the visual line. The latter does not usually pass through the center of the pupil, and in some cases of irregular or displaced pupils, the visual line may not pass through the pupil at all. This is of practical importance in the adjustment of glasses in aphakia or high myopia. Failure to take this into consideration may introduce a prismatic effect in the lens.

The prismatic effect of the correcting glass is of extreme importance in anisometropia. The cylindric effect of tilting a lens is considered. For instance, glasses placed at the proper angle for reading may introduce astigmatic errors for distance and vice versa.

The effect of the distance between the eye and the correcting glass was considered next. By the use of a simple formula, the size of the retinal image in different forms of ametropia can be determined. The practical significance of this important, as in myopia of high degree the correcting lens should be placed as close to the eye as possible, for by so doing the lens will have the same effect as a stronger lens placed farther away from the eye and, at the same time, the retinal image is enlarged.

In this connection, the advantages of deeply curved lenses is pointed out. In the convex lenses the posterior principal point lies in front of the lens and therefore makes the distance from the eye even greater than from the lens itself, and in the concave lenses the principal point lies behind the lens making the distance from the eye even less than from the lens itself.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

WILLIAM F. C. STEINBUGLER, M.D., *Secretary*

October 21, 1929

ERNEST F. KRUG, M.D., *Chairman*

A CASE OF HYSTERICAL AMBLYOPIA. DR. GUERNSEY FREY.

A woman, aged 52, who consulted Dr. Frey, April 5, 1929, gave the following history: Six days previously, after aiding in extinguishing a fire in her place of business, she noticed that she was blind. There had been no improvement and she was being treated with sedatives. The family and the past personal histories were negative. Examination showed that the patient was obviously blind, as she stumbled over a chair. The pupils reacted to light, but the patient saw nothing, with the exception of a general glare, and cooperation was so poor that the projection of light could not be determined. The fundus examination, as well as tests for malingering, was negative. A diagnosis of hysterical amblyopia was made and a good prognosis was given, the family being advised that vision might return suddenly following another shock, or gradually, as

the result of suggestion and encouragement. On the way home, the patient's husband decided that something should be done at once, and accordingly he stopped in at an airdrome and took his wife for a ride. On her return to earth, vision was completely restored and ophthalmic examination showed the eyes to be normal in every respect.

DEMONSTRATION OF COLORED OPHTHALMIC PHOTOGRAPHY. DR. LAURANCE D. REDWAY.

There has been a great advance in color photography of the eye and it is now possible to take and finish a colored plate in about fifteen minutes, since the light can be carefully calculated and controlled. This is of great importance in clinical teaching, and with adequate plates available, it is possible to have a consecutive record of a case from its inception to discharge and to retain what might in any given case be the chief diagnostic feature. The apparatus necessary for the work is simple, a horizontal microscope and a source of light, the relations of which are permanently fixed, being all that is required. A small pilot light affords sufficient illumination for focusing. The patient is seated as for a slit-lamp examination and the eye is subjected to no light save that of the pilot lamp until the instant of exposure. As the latter takes only about one fiftieth of a second, the eye is practically static, and as the light beam is incident to the pupil at an obtuse angle, the greatest intensity does not fall on the macula. For a short time following the exposure there is a scotoma present. Since the plates are smaller than the normal lantern slide, a kit must be used to adapt the ordinary projection apparatus to this use. The color has remained in the plates over a period of two years and has shown no tendency to fade. Copies can easily be made and the originals safely stored away. With the possibility of permanent reproductions, the facility with which the plates may be copied and the simplicity of making them, there is no good reason why color photography should not be widely adopted as a teaching adjunct and a means of improving the general quality of textbook illustrations.

Dr. Redway showed numerous colored photographs of various conditions of the eye.

DISCUSSION

DR. MARK J. SCHOENBERG: I should like to ask whether it is possible to improve the plates instead of the illumination as there are certain points beyond which one cannot go in the way of illumination.

DR. LAURANCE D. REDWAY: The greatest difference between the Lumière plate and the Agfa plate is that in the latter plate the granules are hexagonal and are pushed very tightly together, so that there are no white interspaces between the contiguous grains. In the Lumière plate the elements are not pressed under such great tension, and in consequence there are more light interspaces between the granules. These are filled in with black granular material, and on the whole the density is greater. The plates are probably susceptible of improvement.

DR. GUERNSEY FREY: In June, 1929, Dr. Morax showed me a few colored stereoscopic plates which he had made. They were very beautiful. I am not familiar with his technic but I believe that he uses a flashlight. Would Dr. Redway care to discuss Dr. Morax's methods and results?

DR. LAURANCE D. REDWAY: The plates made by Morax are exceptionally good. Several were published in the *Annales d'Oculistique* in April, 1929. However, Morax, using the ordinary Druner stereoscopic camera put out by Zeiss, has to use the flashlight as an illuminant. That may be all very well for Dr. Morax, but I should hesitate to advise the use of a flashlight, because the flashlight powder itself is exceedingly variable, even if one uses the same brand all the time. It is hard to control the little particles of magnesium; the flash must be set off fairly close to the subject in order to get the necessary illumination, and whereas it can be used as proved by Morax as an excellent laboratory method, it is not in my opinion feasible from a clinical point of view.

DR. WALTER R. LOEWE: I should like to ask Dr. Redway a question about the photography of the fundus which I have been doing, but before I do so I want to congratulate him on his success as demonstrated by these plates. It is only after one has been doing this work, with its heartbreaking failure—taking hundreds of plates and apparently wasting hours in obtaining a picture which proves to be only fair and on repeating the experiment finding something which is no better than the first—that one can fully appreciate what has been achieved in fundus photography. I want to know what filters were used in the Zeiss apparatus for the pictures of the fundus, the length of exposure necessary, and whether there is any injury to the structures of the eye in prolonging and intensifying this exposure sufficiently to obtain colored photographs.

DR. LAURANCE D. REDWAY: The filter to be used will depend on the carbons used in the arc. If one uses the 9 mm. caesium cored carbons one may photograph without a filter. If the ordinary "white-flame" carbons are used, it may be necessary to use a light yellow filter. If one uses some of the carbons which have a little iron in them and give a bluish light, one may have to use a heavier filter. It is almost impossible to give advice about this, as it depends on the arc emission by which the work is done and also on the amperage of one's lamp. These pictures were all taken with the arc operating at exactly 10 amperes, and it is better to take them without any filter by using carbons yielding a suitable spectrum. The temperature in the light beam is about 350 F. The danger is practically nil. I have used a Bausch and Lomb metal leaf shutter to control the light. The light is on for only a short time and the leaves are closed, except during the short interval of the exposure. I have used that arrangement for more than 2,200 photographs, and never once had it stick or cause the least trouble. There is, of course, danger in unduly prolonging the exposure. The normal for pictures of the fundus is from one-tenth to one-twenty-fifth second.

IRIDOTASIS FOR PRIMARY AND SECONDARY GLAUCOMA. DR. GEORGE H. BELL.

This article will be published in full.

DISCUSSION

DR. ERNEST F. KRUG: What is the longest time a case has been under observation, and what happens to the prolapsed iris?

DR. GEORGE H. BELL: I have watched one case for four years; the iris remains in the limbal wound and acts as a wedge.

DR. ALGERNON B. REESE: It is difficult for us to reconcile this operation of iridotasis with what we try not to do in any other operation, namely, incarceration of the iris. In the latter case we feel that it predisposes to glaucoma, whereas in the former it arrests it. In view of the fact that these two conditions are ostensibly the same, their effects seem to be incongruous and it is interesting to theorize on the explanation. Professor Lindner of Vienna told me this spring that he had done iridotasis 200 times with good results. I asked him to advance his theory in regard to the relation of iridotasis to incarceration of the iris, and it was this: When incarceration of the iris happens the iris is usually only knuckled up between the internal lips of the wound and thus allows the external lips and surface to heal, thereby closing the wound and preventing filtration. At the same time the knuckled iris obliterates or embarrasses the filtration angle over perhaps a third to a fourth of its circumference. These two factors predispose to glaucoma. In the case of iridotasis the iris protrudes through the lips of the wound to beyond the external surface, thereby preventing the lips from healing and allowing filtration. It is true that the angle is embarrassed as with incarceration but the filtering wound more than makes up for this.

DR. E. F. KRUG: It must be borne in mind in this particular operation that the iris is not really traumatized. The iris prolapses without having been injured in any way if the operation is properly performed. Is there actually complete healing of the wound?

DR. GEORGE H. BELL: I try not to get complete healing of the wound. After the first day I massage the wound. I felt as Dr. Reese does before I performed this operation, but the clinical results are entirely different from what I thought they would be. It looked like a non-surgical procedure, but it works out well. An ophthalmologist in Chicago has done iridotasis in more than a hundred cases, and I have done it in fifty without a poor result, except in the acute cases, and these were fulminating cases in which operation had been done in other hospitals. In one instance everything had been done and the vision was lost, so we thought we would try iridotasis, and we have kept the tension down. It is the best operation I have seen. I am not offering this operation as a panacea for all the ills of glaucoma, but one does not have to hesitate as to what kind of eye to use it in. Some surgeons do a trephine operation with a shallow anterior chamber, and others do a La Grange operation with a deep anterior chamber, but we do this operation in every one with glaucoma, and the results have been uniformly good.

DR. CHARLES LITTWIN: At St. Luke's Hospital we have done iridotasis about thirty times with remarkably good results, so that Dr. Wiener has decided that it is the operation of choice, and has abandoned the trephine operation entirely. In one case in which iridectomy was done in one eye several years ago and iridotasis in the other eye about a year ago, the tension in the eye treated by iridotasis is below 15, and in the other about 22. As I said, it is the operation of choice in our service, and there are a few points I want to bring out regarding the technic. We never operate on any patient who has had pilocarpine within five or six days, prior to the operation, because we do not want the iris to be pulled down into the wound. Secondly, after we pull the iris out we take a spatula and spread it out wide to fill the entire wound,

and finally, instead of using the three sutures that Dr. Bell uses, we put in a continuous suture to make it watertight and prevent infection. I have observed these cases during the past year, and there seems to be a bleb between the iris prolapse and the conjunctiva which takes up the excess tension; then the patient is taught to massage the eye daily. We have had 100 per cent success.

DR. G. H. BELL: We always tell the patient to use zinc drops at home. He may first use 1 and then 2 grains of zinc sulphate to an ounce of boric acid twice a day to keep down the chances of infection. We tell the patient to report back for a check up every three months. This is very important, as these patients should be seen from time to time.

THE RELATION OF THE PATHOLOGIC LABORATORY TO CLINICAL OPHTHALMOLOGY. DR. S. HANFORD MCKEE.

The bacteriology of the conjunctiva both in health and in disease was first discussed, and the importance of a knowledge of the microorganisms of the diseased conjunctiva was stressed. It was pointed out that streptococci and pneumococci are best demonstrated by the use of blood agar plates. The great variation in the clinical picture in Morax-Axenfeld conjunctivitis was mentioned, as well as its complications of ulceration of the cornea and absorption iritis. Serpiginous ulcers of the cornea are sometimes due to the diplobacillus, and therefore react to treatment with zinc sulphate. Phagocytosis by the conjunctival epithelial cells was considered and special reference was made to the diplobacillus in this connection. A case was also cited in which gonococci were cultivated from a conjunctiva after thirty-five days of treatment. The trachoma bodies of Halberstaeder and Prowazek were discussed, and also the Noguchi bacillus. In 100 consecutive cases of iritis the etiologic factor was syphilis in 48 per cent; focal infection in 22 per cent; gonorrhea in 6 per cent; tuberculosis in 4 per cent, and trauma in 2 per cent. The cause was not determined in 18 per cent.

The details of a case of primary tuberculosis of the iris were presented, with a description of the histologic appearance of the inflamed lacrimal sac. The pathologic reports in cases of retinitis pigmentosa, retinoblastoma, sarcoma of the choroid and a number of orbital tumors were given, together with a description of three cases of sympathetic ophthalmia with pathologic reports. Cure was effected in the latter cases by large doses of sodium salicylate and 3 per cent atropine.

Parinaud's conjunctivitis and blastomycosis were considered and the pathologic conditions in each were discussed. The address was illustrated by a series of eighty lantern slides.

DISCUSSION

DR. ARNOLD KNAPP: DR. MCKEE has covered many chapters in ophthalmology, and has presented a number of most interesting case histories, illustrating them with reports of the pathologic observations, thus again impressing on us the close relationship between pathology and clinical medicine. I am sure we will take home with us a great deal of information and renewed stimulation. I was particularly interested in the case of endothelioma of the optic nerve sheath, and I want to congratulate Dr. McKee on his successful treatment of a patient with a severe case of sympathetic ophthalmia.

Book Reviews

THE HISTORY AND TRADITIONS OF THE MOORFIELDS EYE HOSPITAL. ONE HUNDRED YEARS OF OPHTHALMIC DISCOVERY AND DEVELOPMENT. By E. TREACHER COLLINS, Consulting Surgeon; Member of the Commitee of Management; Formerly, Clinical Assistant; Junior and Senior House Surgeon; Curator of the Museum and Librarian; Assistant Surgeon and Surgeon. Price, 12 shillings, 6 pence. With 27 plates. London: H. K. Lewis & Company, 1929.

Treacher Collins has written this valuable and truly delightful book after an intimate association with Moorfields of more than forty years, during which time he served the institution as clinical assistant, house surgeon, curator of its museum and library, assistant surgeon and surgeon, and finally as consulting surgeon and member of the committee of management, a position in which he is now actively engaged.

In tracing the rise and development of the hospital, he gives the causes which led Saunders, Farre and Battley to create an institution for treatment for diseases of the eye and ear. The subtitle, One Hundred Years of Ophthalmic Discovery and Development, is well chosen, for, as he continues his story, he weaves into it a description of the development of ophthalmic science throughout the world, showing the part which Moorfields played.

Although Saunders is regarded as the actual founder of Moorfields, Farre and Battley were closely associated with him, all three being pupils of Sir Astley Cooper during the latter part of the eighteenth century. Cooper's influence in the founding of the hospital, therefore, must be recorded. Indeed, this famous surgeon actually substituted for Saunders when the latter was ill, and operated in his place.

The London Dispensary for Curing Diseases of the Eye and Ear, as the hospital was first called, was opened to receive patients in 1805, and was situated in Charter House Square. In 1821, the hospital removed to Moorfields, when the name was changed to the London Ophthalmic Infirmary. In 1837, by reason of its receiving continued royal patronage, it was designated as the Royal Ophthalmic Hospital. The name which has clung to it through many years, however, and by which it is best known to its many students, is that of the Moorfields Eye Hospital. According to Collins, "Moorfields" was originally a piece of moorland lying to the north of the old city wall, access to which was obtained through the Moorgate. Early in the seventeenth century it was drained, laid out in walks and planted with trees. For a long time it remained a place of recreation and jollification for the city folk.

The hospital at Moorfields outgrew this site, and in 1899 it was removed to its present situation in the City Road, where its ever increasing work is carried on. It is understood that further alterations in the structure are being planned, and that private rooms will be installed in the near future.

Teaching was begun as early as 1811, and no estimate can be placed on the number of those who have received their instruction in ophthal-

mology in this famous school. Interesting accounts are given of the lives of Tyrrell (Tyrrell's hook) and Travers, both of whom had been pupils of Cooper, of Lawrence, George Critchett, Bowman, Dalrymple, Streatfield, Hulke, Jonathan Hutchinson, George Lawson, Nettleship, Gunn and others. Excellent illustrations accompany the text describing many of these famous men. One of Saunders forms the frontispiece.

The discovery of inhalation anesthesia was a great step forward in surgical intervention of the eye, and when the dangers of vomiting after the administration had been lessened, many new operative procedures were invented and many old methods improved. The technic of tenotomy by Critchett, and the treatment for lacrimal diseases by Bowman, were among these. An account is given of the interesting but dangerous procedure of "iridesis" as devised by Critchett, and practiced extensively by him and Bowman, but relinquished after a time on account of its giving rise to sympathetic ophthalmia in some cases.

The meeting in London, in 1851, of Graefe, Donders and Bowman is described at considerable length. Collins narrates the following: "George Critchett, who was an admirable French Scholar, frequently attended the meetings of the International Ophthalmological Congress, and his son Sir Anderson, was fond of relating how at one of its meetings held in Paris in 1867 he performed the operation of extraction of cataract, before the assembled Congress, on the two eyes of a patient, using his right hand for the one eye and his left for the other. So great was his dexterity that at the conclusion of the operation, Graefe who was presiding at the Congress, threw his arms around his neck and kissed him on both cheeks."

In 1859, Critchett and Bowman extended their clinical instruction at Moorfields by giving a systematic three months' course of lectures on ophthalmic surgery, which were attended by medical men from all over the world. Among these may have been David Livingstone, as it had been the charitable custom at Moorfields since 1854 to charge no fee for instruction to missionaries. In consequence of which, many availed themselves of the instruction they could obtain there, and Livingstone may well have been among these.

The two decades from 1850 to 1870 are described as the Golden Age of Ophthalmology. The surgical measures introduced at that period, particularly that of iridectomy by Graefe for glaucoma, the discovery of the ophthalmoscope by Helmholtz and the recognition and correction of various refractive errors during these years, made such a designation permissible.

Visiting Moorfields almost daily, and in intimate association with its staff and personnel for many years, Collins had the opportunity of collecting many anecdotes. Some of these he has recorded. One about Streatfield is cited as follows: "Streatfield was gifted with remarkable manipulative dexterity, and delighted in using his fingers in a way which most people would have regarded as impracticable. There was indeed, something almost acrobatic in his method of operating, and George Critchett used jokingly to remark that he expected one day, on going into the operating theatre, to find Streatfield removing a cataract whilst at the same time he balanced a feather on his nose."

Clinical assistants were first appointed in 1856. This is of special interest to Americans, as many of our countrymen have held this posi-

tion for varying periods of time, retiring with a certificate of Moorfields, adorned with Saunders' portrait at the top.

About the same time the position of Curator and Registrar was created. This proved to be of far-reaching and great scientific import to ophthalmology. Many valuable papers on pathologic subjects based on work done in the laboratory have appeared in the literature, and there a galaxy of famous men have received their training in pathology. Such men as Brailey, Nettleship, Gunn, Collins, Parsons and Lister have held this position. All these men became surgeons to the hospital later, their laboratory work having proved of inestimable service to them in preparation for this important position.

The *Ophthalmic Hospital Reports* appeared about this time and continued until 1916, when they merged with the *Ophthalmic Review* to form the *British Journal of Ophthalmology*. The *"Reports"* contained papers of the greatest value, and were based on the work observed not only in the laboratory, but also by the clinical staff of the hospital. Among the latter was Jonathan Hutchinson's classic on the different forms of inflammation of the eye, consequent on inherited syphilis.

Moorfields is the parent of many hospitals for diseases of the eye in Great Britain and her colonies; two in India were founded more than a century ago. In America, it is directly responsible for two of the leading ophthalmic institutions: the New York Eye and Ear Infirmary, founded in 1820 by Edward Delafield and J. Kearney Rodgers, who had completed their training at Moorfields, and found the necessity of starting a similar hospital at home. In 1824, the Massachusetts Eye and Ear Infirmary was started in Boston, through the initiative of Edward Reynolds, who had just returned from a course of study in London. Many other Americans of note have studied at Moorfields, and it is probable that no other hospital or school of ophthalmology has had as great an influence on ophthalmology in our country.

One will find in the pages of the book that Hughlings Jackson, at one time an assistant to Hutchinson, acknowledges the value of an ophthalmic training to neurologists, "When a physician sees how carefully and precisely ophthalmic surgeons investigate the simplest case of ocular paralysis, he is getting a lesson in exactness, and will be less likely in his own department of practice to deal in such generalities as that a patient's fit 'had all the characters of an ordinary epileptic fit,' and more likely to take pains to describe the convulsion, the place of onset, the march and the range of the spasm."

It is of interest to find recorded that Sir William Osler was about to take up the study of ophthalmology, and went to Moorfields for that purpose, but seeing Buller already well established in the hospital, he gave up the idea, thinking there would not be room in Canada for more than one well trained ophthalmologist.

One will find a description of the development of the Ophthalmological Society of the United Kingdom, established in 1880 out of the Moorfields' Club, an association of Moorfields' men who met at times informally to discuss ophthalmic matters of interest.

The volume concludes with the query, "Do special hospitals justify their existence?" and the reviewer agrees with the author that Moorfields has certainly done so. In defense of his argument, Collins cites

the great advantages which the hospital affords for mass observation, both clinical and pathologic. Certain it is that it has been this great wealth of material which enabled the staff to acquire the experience which led to the publication of so many valuable textbooks and papers on ophthalmic subjects. Certain it is that this mass of clinical material has been the magnet which has attracted students from every land, and has given them the opportunity of observing every phase of ophthalmic disease, and witnessing all forms of operation on the eye.

Not only will all Moorfields' men past and present read this book with absorbing interest, but all those engaged in the practice of ophthalmology will find it a well told tale of the development of that branch of medicine during the past century.

W. C. P.

LE DALTONISME. By P. BLUM and E. SCHAFF. Price, 20 francs. Pp. 132. Paris: Masson & Cie., 1929.

This monograph offers a review of the subject of color blindness, the means of discovering it and the importance of such tests.

The history of the knowledge of the condition is reviewed, Dalton's classic description of his own case being quoted, and the early work of the poet Goethe, who made a thorough study of the subject, is summarized. Apparently, the first work on the subject by a physician and the first recognition of its importance in the railroad service were in 1885 by Wilson of London. (The authors give 1885 as the date of Wilson's work, but mention several other works with earlier dates. The reference to Wilson is not to be found in their bibliography.)

The Swedish physician Holmgren traced a famous railroad wreck and a war-time disaster to color blind operators, and following his work examinations for daltonism were carried out in many railroad companies.

In 1881, Lord Rayleigh discovered the condition of relative color blindness, which has been found to be more frequent than the more striking absolute form.

The theories of color vision are reviewed, none of them, in the authors' opinion, satisfactorily explaining all the phenomena of color blindness. Since the vision of the so-called red-blind person is defective not only for red but also for green, and that of the blue-blind, not only for blue but also for yellow, the authors believe that it is incorrect to speak of protanopia, deuteranopia and tritanopia, which assume, according to Young's theory, the absence of one color sensitive substance. They proposed to call these anomalies simply daltonism for red-green, for green-red, for blue-yellow and for yellow-blue, according to the predominating color defect. These four forms may be absolute or relative.

The various methods of detecting color blindness are described and criticized. The test with Holmgren wools, which is much used in America, the authors believe is unsatisfactory because by presenting such large colored surfaces, it fails to detect defects of color sense in the macular region and misses cases of relative daltonism which are almost as dangerous for the railway service as the absolute forms. The same applies to the methods of Doel, to the test with colored crayons and to Stilling's colored shadows. The tests with isochromatic plates, on which figures or letters are outlined in small colored circles against

a neutral background, have many advantages over these. The form of these plates most in use is that of Stilling. The only objections to these plates are the number of colors used, which confuses some normal observers, and the fact that differences of brightness between the colored dots and the background may allow the figures to be read by some relatively color blind persons. The authors describe a form of chart devised by them which has been in use on the railroads of Alsace-Lorraine for fifteen years. They use the Landolt broken ring, and the figures of playing cards, only one color being used in each plate, which they believe simplifies the test. By means of this test they have found from 3 to 4 per cent absolute and 8 per cent relative daltonism among male applicants for the railroad service, a higher proportion than that found in any previous statistics. Their cases were checked with the anomaloscope, which always confirmed the results obtained with the plates.

The anomaloscope was devised by Nagel to obtain pure spectral colors in tests of color vision. Its use is recognized by the authors as the only exact means of determining the kind and degree of color blindness, and they advise its use as a check on the other methods in all doubtful cases.

It is too complicated and expensive to be practical in the examination of large numbers of men. Isochromatic plates are recommended, either those of Stilling, the Japanese plates or those of the authors being most satisfactory. A method of testing employees who have been shown to have color defects under the conditions of their work, to demonstrate to them their defect and to pick out a few persons with defects so slight that they may be employed in certain positions is described.

An extensive bibliography accompanies the monograph.

S. R. GIFFORD.

Directory of Ophthalmologic and Otolaryngologic Societies *

FOREIGN

Oxford Ophthalmological Congress

Master: Dr. A. B. Cridland, Salisbury House, Chapel Ash, Wolverhampton, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.
Time: July 10, 11 and 12, 1930.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. T. B. Holloway, 1819 Chestnut St., Philadelphia.
Secretary: Dr. William C. Finnoff, Imperial Bldg., Denver.
Place: Detroit. Time: June 23-27, 1930.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
President-Elect: Dr. J. F. Barnhill, Miami Beach, Florida.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Chicago. Time: October, 1930.

American Ophthalmological Society

President: Dr. W. Campbell Posey, Radnor, Pa.
Secretary: Dr. Emory Hill, 501 E. Franklin St., Richmond, Va.
Place: Hot Springs, Va. Time: June 2, 3 and 4, 1930.

LOCAL

Los Angeles County Medical Society, Section on Ophthalmology and Otolaryngology

President: Dr. Harold S. Muckleston, 6422 Hollywood Blvd., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Place: California Lutheran Hospital, Assembly Room. Time: 8 p. m., first Monday of each month.

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Donald H. O'Rourke, 217 Imperial Bldg., Denver.
Place: First floor, Metropolitan Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. Louis S. Greene, 1710 Rhode Island Ave. N. W., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St. N. W. Time: 8 p. m., third Friday of each month.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Atlanta Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. F. McDuffie, 45 Edgewood Ave., Atlanta, Ga.
Secretary: Dr. Leon E. Brawner, 157 Forrest Ave. N. E., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. N. E. Time: 8 p. m., last Thursday of each month.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. George B. Smith, Rome.
Secretary-Treasurer: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta.

The Chicago Ophthalmological Society

President: Dr. Charles G. Darling, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday from October to May.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. A. Van Osdol, 320 N. Meridian St., Indianapolis.
Secretary: Dr. Robert J. Masters, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: December 10 and 11, 1930.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. Robert J. Masters, Hume-Mansur Bldg., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

New Orleans Ophthalmological and Otological Society

President: Dr. W. A. Wagner, Pere Marquette Bldg., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month October to June.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Leo J. Goldbach, 1012 N. Charles St., Baltimore.
Secretary: Dr. Angus Lloyd MacLean, Johns Hopkins Hospital, Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

New England Ophthalmological Society

President: Dr. W. Holbrook Lowell, 82 Commonwealth Ave., Boston.
Secretary-Treasurer: Dr. E. B. Dunphy, 520 Commonwealth Ave., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Parker Heath, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

The Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. N. Spratt, 900 Nicollet Ave., Minneapolis.
Secretary-Treasurer: Dr. J. H. Morse, 80 S. 7th St., Minneapolis.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. L. R. Forgrave, Logan Bldg., St. Joseph, Mo.
Secretary: Dr. A. E. Eubank, Argyle Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

St. Louis Ophthalmic Society

President: Dr. William H. Luedde, Metropolitan Bldg., St. Louis.
Secretary: Dr. Leo L. Mayer, Carleton Bldg., St. Louis.
Place: St. Louis or Washington University Cafeteria.
Time: 6:30 p. m., fourth Friday of each month from October to April, inclusive.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. Charles M. Swab, Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 1615 Howard St., Omaha.
Place: Medical Tea Room. Time: 5:15 and 7:00 p. m., third Wednesday of each month from October to May.

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Frederick J. Wort, 1080 Broad St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Brooklyn Ophthalmological Society

President: Dr. Robert M. Rogers, 680 St. John's Pl., Brooklyn.
Secretary: Dr. William F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday of February, April, May, October and December.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. John P. O'Keefe, 251 State St., Albany.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

Buffalo Ophthalmologic Club

President: Dr. E. E. Blaauw, 190 Ashland Ave., Buffalo.
Secretary-Treasurer: Dr. A. F. Luhr, Medical Arts Bldg., Buffalo.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. E. F. Krug, 12 W. 44th St., New York.
Secretary: Dr. William F. C. Steinbugler, 815 Park Ave., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. A. Vincent Walker, 833 South Ave., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Assn., 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. George H. Rockwell, Physicians Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. George M. Constans, Bismarck.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

CLEVELAND ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Dr. M. Paul Motto, Rose Bldg., Cleveland.
Secretary: Dr. A. D. Ruedemans, Euclid and E. 93d St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

CLEVELAND OPHTHALMOLOGICAL CLUB

Chairman: Dr. W. E. Bruner, Guardian Bldg., Cleveland.
Secretary: Dr. M. P. Motto, Rose Bldg., Cleveland.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Joseph L. McCool, 909 Stevens Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

THE PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

THE PITTSBURGH SLIT-LAMP SOCIETY

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Arthur Jones, 105 N. 8th St., Boise, Idaho.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

COLLEGE OF PHYSICIANS, PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Maxwell Langdon, 1530 Locust St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

THE RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. J. E. Raia, 276 Broadway, Providence, R. I.
Secretary: Dr. F. W. Dimmitt, 195 Thayer St., Providence, R. I.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Fred Hasty, 119 Seventh Ave., Nashville, Tenn.
Secretary: Dr. Fletcher D. Woodward, Box 162, University, Va.
Place: Louisville, Ky. Time: November, 1930.

THE MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. R. J. Warner, 706 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. W. W. Wilkerson, Jr., Bennie-Dillon Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. D. L. Bettison, Medical Arts Bldg., Dallas, Texas.
Executive Secretary: Dr. W. Mood Knowles, Medical Arts Bldg., Dallas, Texas.
Recording Secretary: Dr. L. A. Nelson, 4105 Live Oak, Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. R. Thompson, 510 Main St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, Fort Worth National Bank Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

HOUSTON ACADEMY OF MEDICINE, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. John H. Foster, 1300 Walker Ave., Houston, Texas.
Secretary: Dr. Edward W. Griffey, Houston Clinic, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

THE SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. F. W. Sorrell, Gibbs Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. A. F. Clark, 728 Main Avenue, San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Will Otto Bell, Medical-Dental Bldg., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, Medical-Dental Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. H. Leroy Smith, Ezra Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. H. B. Stone, 303 1st St., S. W., Roanoke.
Secretary-Treasurer: Dr. Fletcher D. Woodward, Box 162, University.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. O. M. Rott, Paulsen Bldg., Spokane, Wash.
Secretary: Dr. C. A. Veasey, Jr., 421 Riverside Ave., Spokane, Wash.

THE MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. L. P. Allen, 145½ Main St., Oshkosh, Wis.
Secretary-Treasurer: Dr. Edward R. Ryan, 425 E. Water St., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.